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THE QUESTION OF MORTALITY IN INFANTS AND CHILDREN IN INDIA, LAYING SPECIAL EMPHASIS ON MEASURES TO CONTROL IT*

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For centuries together mankind has struggled against the forces of evil and darkness, against superstition and ignorance. In that struggle many have gone down fighting, most have thrown down the gauntlet and adopted the attitude of destiny and fatality whilst only a few, a microscopic few, have withstood all hardship, all misery, and tried to bring their fellow creatures out of the mire of ignorance, superstition and filth into the light of knowledge and cleanliness.

Thus it is not very surprising that even in the twentieth century, so little has been undertaken and achieved in India on the question of mortality in infants and children. Although much has been said and written on the subject by various authorities and institutions who work for the welfare and well-being of the new-born still they have not been in a position to tackle this very important national, nay an international problem, in a satisfactory manner. They have not been able to bring about a satisfactory lowering of the infant mortality rate as is needed at the present time, in a world which has advanced far beyond any one's dreams, socially, industrially and economically. The above is found to be true especially for India from the very high infant mortality rate as compared with that in other parts of the world.

The infant mortality rate is calculated in a different manner from that of the general death rate. It is the number of infants that die under one year of age for every 1,000 births per year whereas the general death rate which includes children over one year of age as well, is calculated on the general population. The reason for this is that the exact number of infants born is known by registration which is not possible at present in India as regards the number of infants born or dead. This leads one to think whether registration of births and deaths, specially in India, is done on a sound and proper basis or is defective somewhere.

It is an acceptable fact that deaths are better registered than births. In spite of various means adopted by different authorities and institutions registration is still very defective in all parts of the subcontinent of India. Various reasons may be deduced for the same. First of all, the men who are employed for this job are mostly part-time. They are obtained mostly from amongst the uneducated or partly educated classes who have very little or no idea of responsibility and thoroughness which is essential for a job of this magnitude involving the health and the very life of a nation. Secondly, the public also, because of its secretive and superstitious habits as regards births etc., do not cooperate as desired with the authorities concerned. Thirdly, registration is not strictly compulsory in all the parts of India; and where it is compulsory, there is no proper legislation by which the defaulters could be prosecuted and made to understand their duties and responsibilities as citizens and human beings.

Births escape registration far more easily than deaths (and this is due to the fact that migration of cases from villages etc., occurs to towns and cities) and swell the infant mortality rate of the latter whilst avoiding the registration of births unknowingly or otherwise. Besides the defective registration, another factor for the high mortality amongst infants is that their birth rate is also very high as is seen by the records of the Public Health Commissioner which are 34, 35, 36 birth rate per mille respectively for the years 1934, 1935 and 1936.

Taking into consideration the above facts, we can remedy them, the first by stricter and adequate registration; and the second by a lower birth rate. In these days of stress and strain, of great hardships and stringent economic conditions, the parents have begun to realise their sense of responsibilities to their children in particular and to the country in general; and as they cannot afford to support large families with the meagre means at their disposal, naturally the high birth rate must fall in time. In this connection one may quote Professor Karl Pearson and others who believe and regard the production of unhealthy infants as eugenically unsound and think that infant

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mortality is Nature's way of removing the unfit and the unwanted. This may be true, but in my humble opinion, we should not try to reduce the population by this crude method.

Whatever may be the case, it is a proved fact that our infant mortality rate is very high, especially in these days of civilization, culture and understanding. The Central Advisory Board of Health in their report on Maternity and Child Welfare work in India by a Special Committee (1938) gives the average quinquennial infant mortality in British India as 2·8 times more than that of England.

Means and ratios of infant mortality rates of British India and England and Wales

Mean of	India	England & Wales	Ratio.
1912-16 ..	.. 204	102	2·0
1917-21 ..	.. 217	89	2·4
1922-26 ..	.. 181	73	2·5
1927-31 ..	.. 170	67	2·6
1932-36 ..	.. 173	61	2·8

The average quinquennial infant mortality in British India, in spite of an appreciable fall (as seen from the above table) since 1912, is now 2·8 times that of England and Wales; although during the first quinquennium given above, it was only twice as high. The rate of fall has, therefore, been slower than that of England and Wales.

For the year 1937, the Public Health Commissioner records the infant mortality rate as 161·7 per thousand live births in British India, whereas the Health Officer of Bombay for the same year gives the rate as 245·0 per thousand live births in the city of Bombay. From the report of Lane Claypon (1920) as shown in a table below, it will be seen that the number of deaths under one year of age per 1,000 births in England and Wales for the years 1891-95 was 151 and that for the year 1918 was 97.

Year.	Infant mortality rate.	Birth rate.
1891-95 ..	.. 151	30·5
1896-1900 ..	.. 156	29·3
1901- 05 ..	.. 138	28·2
1906- 10 ..	.. 117	26·3
1913	.. 107	28·0
1916	.. 91	21·6
1917	.. 97	17·8
1918	.. 97	17·7

From this, it appears that the two rates must be closely associated. It is true, but if one sees for a moment one will find that the fall is not parallel; the birth rate having fallen more rapidly than the infant mortality rate. One can hardly realize what they must have done to bring the death rate amongst infants as low as the above. Even for the year 1937, they have done better, and quote the infant mortality rate as low as 59. They must not have left one stone unturned to achieve the result. How many amongst us, being aware of the above, lift up our finger and raise our voice to try and improve the conditions already prevailing in India.

Child mortality is said to be commonly accompanied by a high rate of mortality amongst infants. It was found that though a disease does not kill an infant in its first year of life, it leaves it maimed, and so it is more likely to catch infection and die from it within the first five or ten years of its life. The child mortality (for British India) from 1-10 years of its life during 1936 was 23·4; whereas that of England and Wales during 1934 was 12·1. For the whole period from 0-10 years, the death rate amongst the children in India is four times as high as that in England. Practically half the children born in India, do not survive the fifth year of their lives. Below is given for comparison, in a tabular form, the observations of the Special Committee (1938) of the Central Advisory Board of Health, regarding the mortality rate for children under 10 years at different age periods for British India in 1936 and the corresponding rates for England and Wales in 1934:—

	% of total deaths under 1 year.	% of total deaths during 1-5 years.	% of total deaths during 5-10 years.	% of total deaths.
England & Wales ..	7·3	3·2	1·6	12·1
British India ..	25·4	17·9	5·4	48·8

From the above it is noted also that the child mortality rates at different age periods are from 3-5 times higher in India than in England. For the whole period of 0-10 years, the death rate amongst children in India is 4 times as high as that in England. These above facts should open our eyes and make us realize the appalling conditions prevailing in India as regards the infant mortality rate. Being an eye-opener they should guide us in establishing more and more infant and child welfare centres and clinics in cities, towns and villages, working for the welfare and well-being of the life and soul of the nation. But this idea of reducing infant mortality should not mean that we should wait till the baby is born in this world to face all sorts of vicissitudes, and then to take measures. We should go a step further in our activities of child welfare to reduce the infant mortality rate by looking after the condition and welfare if the creator of the baby, namely, the mother, who bears the burden for many months, by introducing into the activities of child welfare work, the important activity of antenatal care.

The Influence of Antenatal Conditions on Infant Mortality

McCleary (1933) says that antenatal conditions have an important influence on infant mortality, and mentions that this is not a new idea. It had long been thought that the health of the mother before and during pregnancy had a direct influence upon the health of the unborn child. The study of this aspect of infant mortality, was quickened by the publication of Dr. Ballantyne's Manual of Antenanal Pathology and Hygiene as early as 1902. Ballantyne (1902) brought into prominence the important part played by antenatal causes, including parental alcoholism, in swelling the infantile mortality rate and also lessening the birth rate. He also mentions and lays stress on the fact that from the third month onward, the infant is brought into close relation, nay a still closer contact with its mother. The placenta, amongst its other functions, acts also as a filter, and elements of fœtal tissues such as bone, muscle and fœtal fluids as blood, lymph are transmitted by the placenta from the mother's blood to the fœtus.

It does not follow that the elements pass in the same chemical combinations as those in which they are afterwards found in the fœtus. When, however, the filtering function breaks down, then germs, toxins, toxic substances, etc., flow across the fœtus. When this happens, various effects may be produced. The fœtus may die at once from the action of the poison, or it may develop a disease in a form different from that which is found in the mother, or the pregnancy may end in abortion or in the later stages of gestation, in premature births, and the infant may develop the disease after birth and either die or recover from it maimed for life. The other evil effect that the failure of the filter may have on the infant is the production of malformations, deformities and monstrosities.

According to the same writer, there are three ways in which antenatal morbid conditions may influence infantile life and health. The first by the production of abortions; the second by influencing premature births, which tend to swell the infantile death rate; and the third by the production of fatal diseases and deformities. The last factor of disease and deformity may produce various results, *e.g.,* the infant may be very precarious and the deformity such, *e.g.,* harelip, cleft palate may occur may render feeding difficult. Similarly, congenital heart disease increases to a great extent the risk in the development of bronchitis or pneumonia in the infant. Lastly, any grave condtion of the mother, who is the be all and the end all of the infant at this stage of pregnancy, *e.g.,* hyperemesis, etc., interferes seriously with the development of the infant in the uterus and so the infant later on suffers from congenital debility.

Thus the antenatal morbid condition has a great influence, nay a greater significance, on the infant during its intrauterine existence, first as an impregnated ovum, then as an embryo and then as a fœtus. Even after it is born, it has to cope with and adapt itself to different environments and sudden changes which have an exerting effect on its individuality which either disable it throughout its life time or end its existence in the very beginning or the very early period of its life. The greatest infant mortality is seen to occur in the first month of life, especially in the first week of the first month. This is confirmed by the Health Officer's Report for the year 1937, tabulated below:—

Table of deaths amongst infants by age period in 1937.

Age period.	No. of deaths.	Percentage of deaths in age period to the total deaths under 1 year of age.
Under 7 days ..	1850 ..	21·4
1 to 4 weeks	1354 ..	15·6
4 weeks to 6 months ..	2371 ..	27·2
6 months to 12 months ..	3113 ..	35·8
Total ..	8688 ..	100·0

The mortality amongst infants at the end of the first month of life was thus 3204 or 37·0 per cent of the total infant deaths, as seen from the above report.

The question has often been asked, as to the reason of the infant succumbing so early, as in its first month and specially in the first week of the first month of life; but little has been done and very little achieved in answering the solution. I think that, if antenatal work is generalised and given all the importance it so richly deserves, and if adequate and thorough midwifery services are set up everywhere, the number of deaths occurring so early in life would begin to fall; and we would have the satisfaction of achieving an atom of the gigantic task staring us in the face. The above is confirmed by Dr. Syke's statement. His analysis of the infant mortality statistics led him to the conclusion that in order to diminish infant mortality it was necessary, nay essential, "to ameliorate, firstly, the prenatal conditions and secondly, the postnatal conditions, and that during both these periods efforts should be first exhausted upon the mother, before confining the attention to the infant."

In trying to reduce infant mortality rate in India, the greatest stumbling block is that, there is no proper supervision of delivery cases by trained people in many parts of India, even in large towns and cities. Confinement done from olden times by the indigenous *dais* is still in practice, and in full swing, if not so much in cities and towns as in the villages and outlying districts. The idea of antenatal examination is quite strange to them; and they take it as a new-fangled idea of someone's vivid imagination, and a pollution and a sacrilege.

In my yearly rounds for the past few years which gave me an opportunity of visiting about a hundred villages in and around the Bombay Presidency, I have come to the conclusion that the methods adopted by these so called "Barber-Midwives" in delivering cases under their care, are abominable and leave much for real improvement. At one place, a *dai* was actually tearing the thin cervical wall with her long dirty nails, as the head was taking a long time in coming out. This showed that either she had no patience to wait or else she wanted to show the relatives of the patient that she was doing something for the reward she was expecting.

Another incident was that of a multipara (third pregnancy) who was being delivered by an untrained *dai,* just next door to our place in the village where we were staying. It was a breech presentation, and as the *dai* was actually having a tug-of-war with the after-coming head, the mother-in-law of the patient came to call us. On enquiry it was found that the two previous deliveries were also done by the same *dai* and the infants had died soon after birth in both the cases. This time too, if it were not for our presence there in the village at the critical moment, the infant would have been born asphyxiated and would have died soon after birth. At one out-of-the-way village in the district we found the deliveries being conducted by an absolutely blind *dai,* in whom the people of the village had so much faith, that in spite of there being two other trained *dais* nearby in the village, more cases were done by her than the other two. Such being the condition of things in India, it is little wonder that infant mortality rate still remains high.

The Health Officer of Bombay gives in his report (mentioned below) for the year 1937, the figure of mothers dying during child-birth as 165 out of which 49 deaths are from puerperal sepsis, which is the highest figure for any other disease of the puerperal state.

Number	Specific Causes of death	No. of deaths
1.	Antepartum hæmorrhage	7
2.	Postpartum hæmorrhage	23
3.	Toxæmias (Eclampsia, hyperemesis, a.y.a. alb.)	32

4.	Puerperal sepsis (sapræmia, septicæmia, P.A.D., coli infection, etc.) ..	49
5.	Embolism	4
6.	Shock during or immediately after delivery	7
7.	Anæmia of pregnancy	15
8.	Malaria, influenza, pneumonia, T.B., heart diseases, complication of pregnancy ..	nil
9.	Operative interference	1
10.	Rupture of the uterus	4
11.	Puerperal insanity	1
12.	Other causes	22

This shows what havoc is played amongst pregnant mothers by these indigenous *dais*, and the reason for the maintenance of the high infantile mortality rate which latter would become less if the practice of these *dais* were under a stricter control or stopped altogether.

The other factor that plays havoc in the high infant mortality rate is the social custom of keeping a case of confinement with her new-born babe in an absolutely dark and dingy room, where the woman and her baby are not allowed to have the day light for 20-40 days at a stretch. In this wrong and unhealthy environment one could not expect any other result than a poor weakly rickety sort of a child, and a weak and frightfully anæmic mother. If the infant happens to survive in spite of this weak and poor condition, it is liable to get any other disease sooner, and die from it either in its first year or within the first five years of its life. Again if it is fortunate enough and able enough to fight that disease still further and survive, then, it will remain maimed or invalid throughout its life time.

From the above it will be seen that infant morbidity is closely related to infant mortality. These weaklings growing up to an adult age and not being able to discharge their duties as normal citizens become a burden not only to themselves, their parents and families, but ultimately even to their country. This was found to be true during the war of 1914-18, when young men were wanted as recruits, a large number of them were found unfit, suffering from one ailment or the other, the result of the preventable diseases of infancy and childhood. If these defects had been corrected and nipped in the bud, the generation would have turned out to be much healthier and it would have been a real credit to the country.

In order to have an A_1, nation, which is the aim of all the countries of the world at the present time, we must first of all try and find out the various causes of infant morbidity and infant mortality. Besides the predisposing factors such as ignorance, superstition, poverty, overcrowding and religious and social customs there are many more, which owing to lack of proper statistics and records, are very ill-defined. As already stated deaths are not certified regularly by medical practitioners in the different parts of India; and the infant or child may have died without any medical attendance. So, exact causes of infant deaths are not to be had for statistical purposes. Although the death certificates in most cases state the causes of deaths as "diarrhœa", "general weakness", "fevers", "convulsions", etc., still these, being only the symptoms of certain diseases, indicate nothing. The notification of births, if made compulsory within 36 hours of birth, as in England and other countries will go a great way in finding out the proper causes of infantile mortality and consequently in finding out the means for reducing the infantile mortality rate.

It is found to be true that hospitals do not keep the delivery cases for more than 10 days; and sometimes they even discharge them within 7 days, owing to scarcity of beds, etc. These new-born babes are put on the right path of good health and future well-being for the first 10 days when they are in the hospital or are under proper medical supervision; and they begin to deviate from the above path in the remaining 355 days of their first year of life. Is this, because there is no one to guide them or supervise them or is it because of the economic and the environmental conditions at home that react on their health and well-being? The answer is found in the report of the Public Health Commissioner for the year 1938, in which he writes that "Investigation of the clinical causes of death in infancy and childhood is much to be desired, but it must be recognised that accurate diagnosis of the clinical cause of death may be of less moment, from the view point of saving life, than a knowledge of the social, economic and environmental conditions which react on the child's health from the day of its birth."

For investigation purposes, the causes of deaths may be divided according to age-periods. (1) Within the first month. (2) The other 11 months; *i.e.*, (*i*) neonatal and (*ii*) postnatal periods respectively.

Neonatal Deaths and Their Causes

The largest figures for neonatal deaths relate to two main varieties of causes: 1. Infantile debility, malformation, prematurity, etc. 2. Careless midwifery causing infection (particularly due to confinements done by indigenous *dais* or midwives) and birth injuries.

Prematurity and infantile debility—Besides the general illness in the mother, during antenatal period, it has been found that in many cases of prematurity, no exact cause can be traced. Such cases are usually found in primiparas, and here prematurity came into prominence as one of the most important causes of neonatal deaths. This was shown by the steady rise of the mortality rate in England, as 12·8 in 1876 and 19·9 in 1900. This 19·9 per 1000 births in 1900 was out of a total mortality of 154. At first it was attributed to the employment of married women in factories and workshops; but this hypothesis received no support from later investigations.

In Chicago, Bundesen and others (1938) in a series of postmortem examinations on 911 infants, out of which 600 were premature ones and the remaining 311 were full-term infants, find that in 320 premature and 42 full-term ones making a total of 362 infants, no demonstrable pathological condition could be noted.

In India and specially in Bombay, Nerurker (1934) in his report mentions about the steady rise of neonatal deaths from premature births and gives the very significant figures for the years 1901 to 1932.

Cause ..	1905	1910	1915	1901 1920	1906 1921	1911 1922	1916 1923	1924	1925	1926	1927	1928	1929	1930	1931	1932
Prematurity	5·3	8·6	9·5	17·0	24·5	21·7	20·3	24·4	19·7	27·9	21·9	19·0	24·0	21·7	25·8	25·4

The Health Officer of Bombay in his report for the year 1937 records 906 as premature infants out of 8688 infant deaths; and the Central Advisory Board of Health in its report on maternity and child-welfare work in India by a Special Committee give the figures in percentages for prematurity for the three presidencies of Bombay, Bengal and Madras as 16·1, 17·5 and 12·7. Balfour (1930) in her recent investigation in the Urbs Prima in India, on all prematurely born infants and infants with weight below 4 lbs. at birth mentions that out of every 1,000 prematurely born infants, 840 died within the first 3 months. Dr. Christine Thomson's inquiry also confirm the above statement and show that prematurity is a very common factor for still-births and neonatal deaths amongst infants in India.

Similarly, the largest figures (shown in the table below) of the chief causes of mortality in infants in the City of Bombay during the years 1932-36, prepared by the Special Committee of the Central Advisory Board of Health, relate to two groups of causes (1) "debility, malformation and premature birth" and (2) "respiratory diseases". Of these groups the first namely prematurity takes the greatest toll specially within the first month or even the first week of life.

	1932	1933	1934	1935	1936
All Causes	6,298	8,320	8,253	8,455	8,946
1. Small pox	69	717	33	306	214
2. Measles	16	18	27	60	42
3. Malaria	4	9	9	9	3
4. Remittent and undefined fever	147	167	186	146	114
5. Diarrhœa & enteritis ..	280	414	455	425	539
6. Dysentery	27	19	40	41	48
7. Debility, malformations & premature births ..	2,685	3,019	3,384	3,280	3,651
8. Respiratory diseases ..	2,217	2,828	2,983	3,039	3,174
9. Convulsions	422	594	562	563	683
10. Other causes	431	535	574	586	478

The above statement is verified by the following figures taken from the Bombay City Health Report for the year 1936. This report also shows that the percentages for the first group, namely, prematurity, etc., decline progressively as the infant grows older whilst those of the second group, namely, respiratory diseases, etc., steadily go on increasing.

Groups	Age Periods			
	Under 1 wk.	1 to 4 wk.	4 wk. to 6 mo.	6 to 12 mo.
I. Prematurity, infantile debility, malformation etc.	88·3	75·6	22·4	4·7
II. Respiratory diseases etc.	1·0	3·8	46·7	67·2

Careless midwifery causing infection and birth-injuries— This is another most important cause of neonatal deaths amongst infants, specially so in India; because of the old-practised methods of confinements done by "Barber-Midwives" as they are called or indigenous midwives or *dais*. These untrained midwives or *dais* are very largely responsible for the maintenance of the high infant mortality rate; because of their unskilful handling of confinement cases which come under their care owing to the ignorance and superstition of the teeming masses of India. This practice by *dais* still goes on, nay even thrives in most parts of India, as the profession has been handed down from one generation to another owing to the poor populace having more faith in their old *dais*, rather than in the trained midwives.

The Lady Wilson Village Maternity Association, which was giving all the facilities for training these *dais* in the Bombay Presidency had to close; because more deaths from infection and birth injuries were found to occur at the hands of these semi-trained *dais*, who left their good training and cleanly habits behind and followed the old trends and habits handed down to them by the seniors of their profession as soon as they were out of the control, supervision and guidance of the association. The *dais* trained by the above association were provided with a simple outfit which consisted of an aluminium bowl with a lid which served the double purpose of keeping the whole outfit in it, as well as that of sterilizing the pair of scissors and cord ligatures etc. A soap box with a cake of antiseptic soap were also furnished with the above.

In spite of good instruction, advice and care, some of these *dais* were found by me, in one of my village visits, using the empty soap-box as a snuff-box and the aluminium bowl for keeping onions, chillies and other vegetables. It was a thoroughly disappointing state of affairs, and I think that, as this profession goes on in certain families as an heirloom, from one generation to the other, these *dais* will rather prefer and follow the advice of their old mothers or mothers-in-law, rather than the good and careful training given them; with the result that infant mortality rate will remain high in India.

Also the deaths due to sepsis and tetanus will continue till the *dais* practice exists amongst us. The Health Officer of Bombay, gives the figure for tetanus as 121 and birth injuries as 38 out of 8688 infant deaths in the year 1937. Before proceeding to the causes of postnatal deaths it would not be out of place to mention here about the articles in the *Lancet*, May, 1940, by McNeil. In a series of articles about infant mortality he says that the neonatal deaths as determined by necropsy are divided into three main groups, such as, (1) intracranial hæmorrhage; (2) infections; (3) a miscellaneous group made up of asphyxia, congenital defects and other conditions; and the three groups are approximately equal in the production of infant mortality rate. He also gives, after studying the neonatal deaths in a large Maternity Hospital, the causes as those during the first two weeks after birth and those in the second fortnight. In the former he mentions congenital defects, asphyxia, intracranial hæmorrhage and infection; and behind some of these again, important predisposing conditions such as maternal disease in pregnancy, difficulty in labour and the most important of all prematurity.

(*To be concluded*)

CARRIERS

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Introduction

In any infection the end result may either be the patient's death, or the extinction of the infection or the survival of both the patient and the infecting agent. In the last condition the patient continues to harbour the organisms in his body and is usually called a carrier. A carrier is a human being who although not ill, is infected with and capable of disseminating the causal microorganism or parasite of some disease. He need not have been a patient as one may become a carrier simply by coming in contact with a patient.

In 1889-90 it was observed that diphtheria bacilli might be found in the throat of a healthy person. The search for, and discovery of carriers was immensely stimulated by Frosch's suggestion (1903-4), and Drigalski's proof (1904), that typhoid bacilli could be propagated in the human body and discharged long after recovery from the typhoid fever. Prior to these observations Koch had urged the proposition that an ambulant convalescent was the chief disseminator of the disease. Koch's anti-typhoid campaign and Conradi's researches (1909) followed by similar works in Great Britain by Ledingham (1912) and elsewhere, and the recognition of paratyphoid carriers by Conradi (1906), established the predominating importance of carriers in the enteric group of diseases. Smith (1934) pointed out with emphasis that successful parasitism, from the point of view of the invader, involves a mutual tolerance of the host and the parasite, so that both survive with a minimum of inconvenience to either. This state of equilibrium, approaching symbiosis, is responsible for the more dangerous type of carriers. It appears to be the final stage in the evolution of successful parasitism. Carriers constitute the most potent source of danger to a community.

Carriers in Different Diseases

Typhoid Fever—The subject of carriers in typhoid fever has been reviewed exhaustively by Browning and his collaborators (1933), and what follows is based largely upon their monograph.

Typhoid carriers may be either temporary—those who excrete the organisms for short periods during or after convalescence or chronic, if this period extends to six months after the acute attack. If the excretion of the organisms extends to a year it is unlikely to clear up spontaneously and will become permanent.

Carriers are considered under two primary groups as 'fæcal' (intestinal) and 'urinary' excretors. Occasionally the organisms may be discharged from abscesses or sinuses. The fæcal excretors may be further divided into (i) biliary carriers and (ii) true intestinal carriers. The latter are much more uncommon than the former, but, as Browning points out that their recognition is important since a proportion of failures to obtain cure by cholecystotomy may lead by elimination to the diagnosis of true intestinal carrier. Children rarely become chronic carriers.

Urinary excretors are much less common than fæcal excretors. Indeed a person may go through a typhoid infection and yet manifest no symptoms of the disease. Such a person may become a carrier though possessing no knowledge of the fact that he has suffered from this infection. They are particularly dangerous when they happened to be engaged in handling food or milk. Many localised epidemics have been traced to such carriers.

The Journal of American Medical Association (1938, 111:2023) reports the death at the age of 70 of Miss Mary Mallon, commonly known as "Typhoid Mary" who was the best known example of a typhoid carrier. During her employment as cook in various households and institutions she gave rise to at least ten outbreaks with 51 cases, and probably more until she was interned in a New York hospital for the last twenty-four years of her life. A full account of her was published by Soper (1919).

The detection of carriers rests entirely with the pathologist. Culture of the stool, urine and detection of Vi-agglutinins in the serum are to be employed for this purpose.

Bacillary Dysentery—Manson-Bahr (1939) classifies the carriers of bacillary dysentery as healthy, convalescent, relapsing and chronic. The *healthy carrier* is one who passes dysentery bacilli along with fæces without having suffered from an acute attack of the disease. Clinically the person is healthy. This group is rare, this method of spread is also not of great importance for since the cases reported by Kruse (1900), Conradi (1903) and Mayer (1911) little work has appeared, except that by Vazquez-Colet (1925) in the Philippines. The organisms may, however, appear in the fæces in an intermittent manner, as is shown by Verzár and Weszeczky (1916). As regards the bacteriology in such carriers the majority harbours Flexner's bacilli. The *convalescent carriers* constitute the largest bulk. A convalescent carrier is a person who, subsquently to an attack of bacillary dysentery continues to pass viable bacilli in the fæces, usually together with some mucus and blood. Saquépée (1910) considers that the formes frustes, the slight and clinically almost unrecognizable forms of the disease, play an important part in its spread. Convalescent carriers are always the most important source of small epidemics. Thus Stitt (1922) quotes an instance of a small epidemic of Shiga type recorded by Friedmann (1913); an infected soldier returned to barracks after furlough, and this resulted in 86 cases in the man's regiment, 49 of which belonged to his own squadron. Mild and unrecognised cases are also important factors in the spread of epidemics. The *relapsing carrier* is one by whom dysentery bacilli are passed in the fæces owing to renewed activity of the organisms in the intestinal canal. Such cases have been reported by Hudson and Manson-Bahr (1939). The *chronic carrier* may be defined as one who is incompletely cured of the disease in a clinical sense, and continues to pass dysentery bacilli in his fæces. According to Fletcher and Mackinnon (1918) Shiga-bacillus carriers were rare in a group of 935 war convalescents, only 1·39 per cent, while 61 were Flexner carriers.

The carrier state in bacillary dysentery does not as a rule persist for any great length of time. According to Perry (1925) the carrier state usually persisted from four to six months, and

at the end of a year only four per cent were still carrying dysentery bacilli; in the case of Flexner infection, it was higher, *viz.*, seven per cent. The carriers can be detected from the history of having had suffered from dysentery or suffering from it at the time of examination, microscopical and bacteriological examination of stool, and agglutination of their serum with the homologous organisms.

Cholera—The cholera vibrios are passed in enormous numbers in the fæces during the early part of the disease. They usually disappear after from the fourth to the fourteenth day, but may remain for one or two months. These are convalescent carriers. Chronic carriers are rare. Healthy or passive carriers occur and are an important means of spreading the infection; they may also develop the disease. Mclaughlin (1908) found numerous carriers in epidemic centres in the Philippine islands—6 to 7 per cent among healthy individuals living in infected neighbourhoods in Manila—but rare in neighbourhoods having few cases. Carriers are the principal factor in the spread of epidemic cholera and in keeping the infection alive. Many carriers continue to excrete the vibrios for a considerable time after recovery, sometimes for two months but rarely more than 20 or 30 days. It was estimated in the Naples epidemic in 1911, that 90% of the cases were due to direct contact with patients or healthy carriers; while in the Colombo outbreak in 1926, of 442 contacts examined 10% were found to be carriers of V. cholera. Fortunately the bacilli usually completely disappear from the stools of convalescent patients in 4 or 5 days, but Grieg (1942) found virulent organisms in the stools of 36% of the patients shortly before their discharge from hospital during an epidemic in Puri; so that the disease was rapidly disseminated by the convalescents travelling by rail to the central provinces several hundred miles away. In Mesopotamia, Mackie and Trasler found that only 7·2% of cholera patients remained carriers for more than 10 days after an attack, and only 2 out of several hundreds continued to be so upto 5 and 7 weeks. Persistent carriers happily do not occur to the same extent as after typhoid fever. The same observers found that 2% of the healthy contacts with cholera patients became carriers without having suffered from an attack of cholera.

For the detection of the carriers specimens are best obtained by administering a saline cathartic or by using a rectal tube with "eyes" cut into it. Several methods of investigation, based upon the facility with which the vibrio grows upon Dunham's alkaline peptone solution are employed. Particles of fæces are planted in this medium and subsequently examined for comma-shaped microorganisms. If found, the diagnosis is presumptive. Pure cultures should then be made and studied for agglutination.

Diphtheria—It must be remembered that the bacilli may remain in the throat long after the patient is himself quite well, and hence the risk of contagion remains. Sometimes a period of several weeks elapses before the patient is free but 50% lose them at the same time as the membrane, not more than 7% retain them for one month, and more than 1 or 2% for three months. Diphtheria is a 'contact' infection, the common mode of being by droplet spray. Overcrowding, especially in bedrooms and dormitories, facilitates infection in this manner. All carriers are not equally effective distributors of the disease, intimate contact and addiction to such habits as kissing, sneezing, spitting and pencil suckling are of importance in the spread of the disease. It is believed that the nasal carrier is an especial source of danger in this respect. Throat and nose cultures from all exposed persons and from all persons in an institutional epidemic should be made. All positive carriers based upon morphological diagnosis should be quarantined. At a later date when time permits, the carriers of avirulent bacilli may be released. It is sometimes necessary to go through a camp or school twice, thrice or oftener to detect all the carriers or to discover new ones. Most of the dangerous carriers even in a metropolitan city can be discovered by taking cultures from the persons associated with cases. Mere presence of diphtheria bacilli in persons does not necessarily mean that they are virulent. They must be tested for the virulence on the guinea pig.

Cerebrospinal fever—Epidemics of cerebrospinal fever are marked by several features peculiar to the disease. For a long time these features baffled explanation until the existence of carriers became recognised and supplied the solution to the epidemiological poblem. Amongst these curious features may be mentioned the erratic nature of the outbreaks, the inability to trace the connection between one epidemic and the other, the relative or even total escape of certain localities close to others in which the disease was rife, and the small proportion of persons affected in any one district. In closed communities, such as camps and especially in times of war, the disease finds a suitable soil. In such communities the majority of individuals may be carriers and only a small minority may be infected. In cities the morbidity is not more than 1 or 2 per 10,000 of the population. The explanation lies in the fact that the receptivity of the throat is high, while the susceptibility of the meninges is low. The carriers, therefore, always greatly outnumber the patients. It has been found that a carrier epidemic always appears in advance of and in association with a case epidemic. In the mode of infection of the meninges the first stage is the infection of the upper respiratory tract, in particular the upper part of the nasopharynx. If the process stops at this stage, the result is the production of a carrier.

Pneumonia—The American observers have shown that 40% of contacts with cases of pneumonia belonging to types I & II may harbour the corresponding organism for an average period of 23 days and that they may develop pneumonia from it. They have further shown that a convalescent patient may carry pathogenic pneumococci in his mouth for as long as 90 days from the onset of the disease. So we see that pneumonia is capable of being spread by carriers, and by convalescent patients.

Scarlet fever—Intermediary carriers have on occasions spread the disease without otherwise showing signs of infection. On rare occasions, convalescents although apparently healthy, have for months remained capable of transmitting the fever. It appears that the infectivity of carriers is intermittent. Convalescent carriers are responsible for something less than 3% of the cases traced in fever hospitals.

Brucelliasis—Out of 525 dock hands examined by Shaw (1941) in Malta, 79 gave positive agglutination reactions. whilst 9 out of 22 of the specially tested showed the organisms

either in the blood or urine or both. Such cases constitute human carriers.

Whooping Cough—On rare occasions convalescents are said to have acted as carriers.

Typhus—Carriers of typhus are always verminous persons. Nicolle (1924) described the 'infectiones inapparentes' with relation to typhus fever. It is a stage earlier in the evolution of successful parasitism.

Acute Anterior Poliomyelitis—Carriers of the disease, who do not themselves suffer, but from whose respiratory passages the virus of the disease can be recovered, are probably common, and these carriers are perhaps the sole vectors in the propagation of the disease.

Epidemic Encephalitis—In its etiology no example of case-to-case infection is available. It is presumed that infection takes place from human vectors alone and by droplet infection. Economo and Levaditi (1937) consider that the healthy carriers and those in the presymptomatic stage of the disease are capable of transmitting the infection.

Helminths—Carriers of helminths (ascaris, hookworm, trematodes, & schistosomes) are abundant. In the United Provinces according to Chandler (1939) 57-93%, and according to Megaw (1939) 20-99% of population are infected with ankylostoma duodenale. Mild infection does not do much harm to patients but undoubtedly constitutes a great source of danger to the community when they act as carriers. Promiscuous habit of passing stools on fields is the cause of infecting the people. The economic loss to India caused by helminthic infection is tremendous and probably stands third in this respect next to tuberculosis and malaria. Helminthic infection *per se* does not kill people but the concomittant rundown in health with extreme degree of anæmia makes them easy prey to other diseases. The villagers must be taught the mode of such infection and above all the habit of defæcating on open fields must be stopped and some sort of foot wear must be used by them.

Amœbiasis—It has been suggested [Kuenen and Swellengrebel (1913), Woodcock (1915) and Brug (1922)] that occasionally E. histolytica may live in a state of commensalism, feeding on the bacterial content of the gut, and applied to the surface of the mucosa instead of burrowing into the deeper tissues. The knowledge that the amœbæ will ingest, in the culture tube, bacterial organisms certainly lends support to this view, but, until further and more conclusive evidence is adduced, E. histolytica must remain a definitely pathogenic agent and a tissue parasite. The carrier question is of vital importance in the spread of the disease. It is important to remember that the term "carrier" is used in a somewhat peculiar sense in reference to amœbiasis. The term has a definite and special meaning, which differs from that with which it is often used by bacteriologists. It would be remembered if the host "carries" anything, it is obviously the amœbæ in his gut wall. He is therefore, correctly called a carrier of E. histolytica," or an "amœbic carrier"; but to call him a "cyst carrier"—as is so often done—is obviously inappropriate. The carriers have a great practical importance in the etiology of amœbiasis as they are the people who are responsible for the spread of the disease. A carrier can be recognised by the examination of his or her stool preferably after a saline purgative.

Comments

This group of diseases is the most prevalent and damaging of all the infections to which flesh is heir. Man is the source and fountainhead of these infections. Each case is a focus of infection—a potential epidemic. The group is properly called contact diseases; they are contagious but in varying degree, limiting the number of persons who come in contact with the patient will lessen the number of cases of carriers. These infections are spread not only by mild and missed cases, but are often particularly communicable during the early stages before the diagnosis is clear.

Endemicity of a disease is maintained by carriers. The outbreak of epidemics is actually composed of a carrier epidemic and a clinical epidemic. From epidemic and endemic outbreaks the carrier-rate rises. The rise in carrier-rate is only a forerunner of the future epidemic and endemic outbreak of diseases which take away millions of people and leave still greater number of millions with maimed, disordered and worn out bodies which entail a tremendous economic loss upon the country. There is little doubt that from the epidemic cases, or from a smouldering infection fresh epidemics light up. By whatever means the machinery is set in motion, it is clear that a necessary precedent to an epidemic is a considerable collection of carriers whence there is the transference of the causal organism to a fresh host. Thus we see that the carriers play a conspicuous role in the perpetuation of a vicious circle of endemic and epidemic outbreak of diseases. The education of the public concerning certain sanitary habits should, therefore, be carried out. These include the danger of spitting promiscuously and of kissing; the proper care to be exercised in sneezing and coughing; the peril in the common drinking cups, the roller towel; and the habit of placing unnecessary things in the mouth, especially the fingers. Restaurants, hotels and soda fountains should be required to scald all glasses, cups, spoons etc., every time they are used. Lynch and Cumming emphasize the importance of the sputum-borne diseases through hand-to-mouth infection and the use of scalding water to wash mess kits and dishes in hotels, restaurants etc. War against disease demands the greatest attention in getting rid of the carriers. If we can prevent people from becoming carriers, we shall be able to get rid of numerous infectious diseases. With the march of progress in the knowledge of medicine many a specific drug has been found out, but there can be no better remedy than the prevention of diseases. Every word of the old English adage, 'prevention is better than cure' is worth in gold. This is in essence the whole argument for the culture of healthy principles of personal and social hygiene by which we can prevent many diseases which are undoubtedly preventable.

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(*Continued at the foot of the page 191*)

INDIAN MEDICINE—AYURVEDA

VAYU

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During the last few years a vast amount of literature has grown regarding the proper interpretation of the *Tridosa* theory, the basic principles of Ayurveda. A critical review of this literature at once reveals that, practically speaking, no two persons agree in their essential interpretation of the *Tridosa* theory. From the point of view of this wide divergence of opinion as detailed in the literature as also from that obtained during personal discussion with eminent Ayurvedic practitioners, even a casual observer is impressed that the proper understanding of the *Tridosa* theory is far from being achieved. This is the only plea for further addition of the following lines to the already abundant literature in consideration of the fact that this might clarify the problem effectively instead of making the confusion more confusing.

For the purpose of the present study, the material available in the *Samhitas* alone has mainly been utilised. This selection is done with a view to get as much of the original material elaborated by the originators of the *Tridosa* theory as can be availed of. One must also take into account that even these original works as extant to the present day, show good amount of interpolations, omissions and misappropriations. A scrutiny of this available material shows evidence of a very rich collection of facts which on closer study reveals the inherent key to the understanding of the *Tridosa* theory.

A Study of the Internal Evidence

The internal evidence as found in the *Samhitas*, reveals that they can be grouped under the following main heads:—

1. Functions of Normal *Vayu* **(प्रकृतिस्थ वायोः कर्म्मांणि)**

वायुस्तन्त्रयन्त्रधरः,.....हेतुमूतः ।—*C. S. 12|8.
उत्साहोच्छ्वासनिश्वास.....कर्म विकारजम् ।—C. S. 18|42.
सर्व्वाहि चेष्टा.....मतः ।—C. S. 17|64.
प्रस्पन्दनोद्वहन.....धारयति ।—S. S. 15|3.

2. Five Divisions of *Vayu* and their Functions

(पञ्चविधवायोर्नामानि सामान्य कर्म्मानि च)

प्राणोदान समानाख्य.....तैरेषाामयः ।—C. C. 28|3-4.
वायुर्योवक्त्र सञ्चारी.....गर्भार्त्तवास्यधः ।—S. N. 1|7-11.

3. Functions of Abnormal *Vayu* **(प्रकुपितस्य वायोः कर्म्मादिकम्)**

सङ्कोचपव्वांनां.....कुपितोऽनिलः ।—C. C. 28|8.
सूंसव्यासवधस्याप.....कर्म्मानि वायो ।—A. H. S. 12|46-47.
स्त्रंसभ्रंसव्यासङ्गभेदः.....वायोः कर्म्मानि ।—C. S. 20|10.

4. Symptoms of *Vayu* covered by *Pitta, Kapha*, etc.

(आवृत वायोर्लक्षणम्)

कर्म्मौजोवलवर्णानां.....उदानेनावृते व्याने ।—C. C. 28|67-68.
लिङ्गंपित्तावृते.....वर्च्चसात्वावृतेऽनिले ।—C. C. 28|21.

5. Functions of *Vayu* according to different locations

(स्थानभेदेन वायोर्लक्षणम्)

तत्रकोष्ठाश्रितेदुष्टे.....प्रवृत्तिः सवेदना ।—C. C. 28|22-34.
अतऊर्जप्रवक्ष्यामि.....शुक्रगेऽनिले ।—S. N. 1|13-24.

6. Eighty Kinds of *Vayu* affections **(अशितिःवातविकाराः)**

नखभेदश्चविपादिकाच.....व्याख्याताः । —C. S. 20|9.

A perusal of the above passages shows that the functions and symptoms attributed to *Vayu* alone are already too numerous. It shows that *Vayu* alone is responsible for the most important functions of the body and at least 313 individual symptoms.

It is striking that a scrutiny of these functions and individual symptoms at once reveals that all of them are the evidence of functions of the nervous system of our body. This will be more apparent when they are arranged under different headings including the different functions of the nervous system.

The different functions of the nervous system may be classified under the following heads and one and all of the above quoted functions and symptoms can be very appropriately arranged under each group as will appear in the table detailed hereafter.

The nervous system consists of 2 main divisions: 1. Cerebrospinal, including brain, spinal cord, cranial and spinal nerves; 2. autonomic, an independent system. Of these two systems the cerebrospinal system regulates the mental functions, functions of the special senses, all locomotions and all sensations of the body which will all disappear as soon as the cerebrospinal system is paralysed; while the functions maintained by the autonomic nervous system, *e.g.*, the circulation, respiration, process of child birth, swallowing, digestion, assimilation, fertilization, being autonomic will go on automatically when the cerebrospinal system is paralysed.

These two systems, the cerebrospinal and the autonomic very often coordinate and although the autonomic nervous system is termed autonomic and is thus dependant on no other nervous system, still it acts in coordination with the peripheral nerves originated from the cerebrospinal system, *e.g.*, in defecation, micturition, parturition etc. This is shown in figure 1.

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* For the purpose of economy of space, these passages are not quoted here in full. They are indicated only with the words forming the beginning and end.

In the references from the *Samhitas* the following abbreviations are used. The first letter, C, S or AH indicates *Caraka Samhita, Susruta Samhita* or *Astangahridaya Samhita* respectively. The second letter S, C, B, Sa or N indicates *Sutrasthanam, Cikitshasthanam, Bimanasthanam, Sarirasthanam*, or *Nidanasthanam* respectively. The first figure indicates the number of the chapter and the last figure the number of the paragraph.

In the figure it will be noted that the spinal nerves are very stout and thick while the fibres of the autonomic system are very thin and fine. This can be explained by using a simile if the spinal nerves are compared with the telephone system and the autonomic nervous system as the electric street supply.

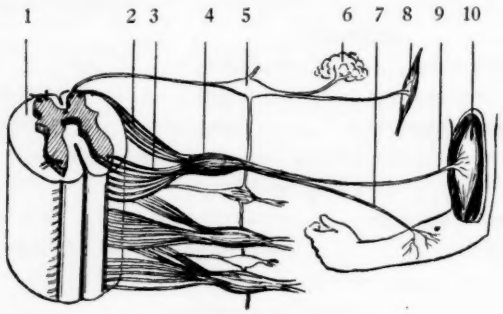

FIG. 1. Showing the relation of nerves and autonomic nervous system.

1. Spinal cord, 2. Anterior nerve roots, (motor), 3. Posterior nerve roots (Sensory), 4. Spinal ganglion, 5. Chain of Autonomic nervous system (*Pingala* in the right side), 6. Secretory gland, 7. Sensory nerve, 8. Involuntary muscle, 9. Motor nerve, 10. Voluntary muscle.

In the telephone system there are always a pair of wires communicating the exchange and the distant telephone. Supposing there are 10,000 telephone connections there must be 20,000 wires running between the exchange and the telephone apparatus. This is the same what is found with the spinal nerves. From every muscle and the skin surface a separate and distinct fibre runs to the spinal cord. According to the number of such fibres the size of the nerve becomes thinner or thicker. Thus the sciatic nerve is about the thickness of the little finger. In the electric street supply, on the other hand, only 2 wires are required to run between the power house and the house connection, the thickness of the wire depending on the load it has got to carry. As in practice the load is relieved by having secondary power stations so that the actual individual house connection is maintained by thinner wires. The same thing takes place in the autonomic nervous system. The main lines do not require thick nerve fibres and the ganglia in the chain of the sympathetic system act as the secondary power houses in the simile. The sympathetic fibres, particularly those supplying the viscera and organs, are necessarily very fine in comparison to the spinal nerves.

The modern knowledge on the autonomic nervous system is mainly due to Langley who published his monograph in 1921. It is surprising that while the knowledge on this very important system, rather the most vital one, has been acquired only during the last 20-30 years, the originators of the *Tridosa* theory came to know of this system in a remarkably accurate way and that in minute details for more than 4000 years. In the words of Langley, "the progress of this system has been continuous for at least the last 250 years, though at times it has progressed but slowly." Still this period of 250 years come to insignificance in comparison to the hoary antiquity of the origin of the *Tridosa* theory which, we shall presently see, mainly deals with the manifestations of this system. Even the *tantric* school made a special study of this subject long before Langley's calculation of 250 years.

AUTONOMIC NERVOUS SYSTEM

The autonomic nervous system is so-called, because of its independence in regulating very important functions of the body, *e.g.*, the movement of the heart, respiration, secretion, etc., and all these, with very rare exceptions, are entirely outside the influence of will. This is differentiated from voluntary movements, which are produced by will. Involuntary movements are put into two categories. First, those, *e.g.*, movements of the respiratory muscles during sleep, which could also be produced by will, and secondly those, *e.g.*, movements of the heart and intestines over which the will has no control, excluding, of course, the supernatural feats demonstrated by the *Yogis*. Ordinarily the functions of the cerebrospinal nervous system are evidenced by voluntary movement, appreciation of sensations, etc., but all the normal functions of the autonomic nervous system are carried on without any effort on the part of the individual and that, again, completely unconsciously. Some amount of visceral reactions, *e.g.*, thirst, hunger, need for visceral evacuation are felt normally and only in abnormal conditions, visceral reactions, *e.g.*, colic, cardiac pain, etc., are perceived. Although this part of the nervous system is beyond the influence of will still it shows a great susceptibility to emotional influences (*e.g.*, fear, rage, grief, joy and other mental excitement) as seen in cases of emotional blushing, sweating, tears, increased heart rate, change in blood pressure, etc.

Anatomy of the Autonomic Nervous System—Any one dissecting the human body is well familiar with two strings of nerves, one on each side of the spinal column, with swellings like knots appearing at intervals. The one on the right is called *Pingala* and that on the left *Ida*. These strings, start from the neck and extend up to the end of the spinal column. The knots in these strings, called ganglia, in the first place, are set more or less regularly in the form of one ganglion to each spinal nerve root, *i.e.*, corresponding to the nerve coming out by the side of each vertebra. Secondly in several places these ganglia appear to form bigger knots. Thus appear in the neck two ganglia named superior cervical ganglion and the inferior cervical ganglion united by the cervical sympathetic trunk. In the thorax, appears the "stellate" ganglion; in the abdomen the semilunar ganglion, and the superior mesenteric and inferior mesentric ganglia. In the pelvis, the pelvic ganglion is seen.

In addition to these groups of ganglia found in connection with the spinal column, nerve fibres of the autonomic system also exist in the brain. These are found in the form of ciliary ganglion in the orbit influencing the action of the pupil of the eyes, submaxillary ganglion influencing the secretion of saliva, and other fibres which run to the œsophagus, stomach and intestines as also the inhibitory fibres to the heart.

Functions of the Autonomic Nervous System—It is found that all the functions of the system can be grouped under two

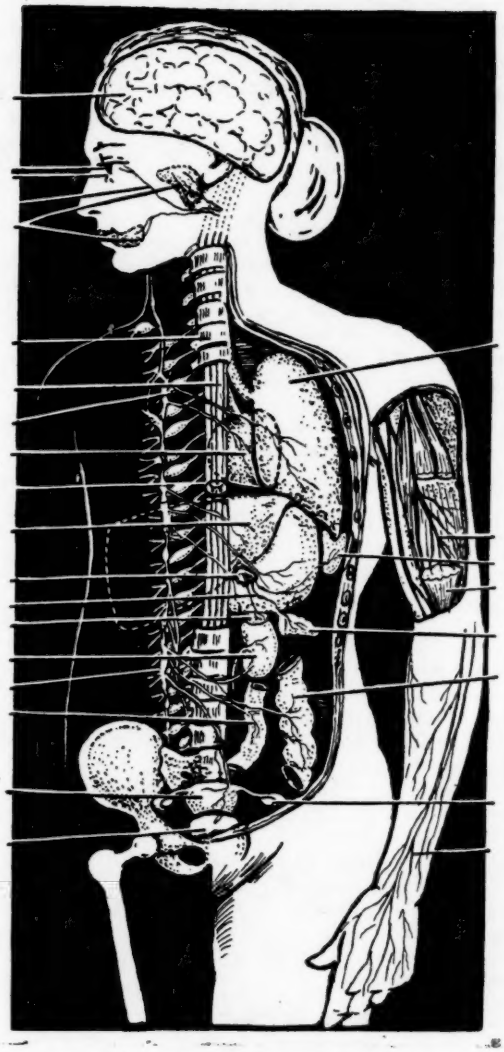

FIG. 2. Showing the anatomy of the autonomic nervous system

1. Brain, 2. Pupil of the eye, 3. Lower eyelid, 4. Nerve fibres to pupil and lachrymal gland, 5. Salivary glands, 6. Spine, 7. Spinal cord, 8. Chain of right Autonomic Nervous System (*Pingala*), 9. Cardiac plexus, 10. Cut end of Spinal cord, 11, Liver, 12. Cœliac ganglion, 13. Fibres to pancreas, 14. Suprarenal, 15. Kidney, 16. Inferior mesenteric ganglion, 17. Small intestine, 18. Uterus, 19. Bladder, 20. Left lung, 21. Nerves to the muscles, 22. Spleen, 23. Muscle, 24. Pancreas, 25. Colon, 26. Ovary, 27. Sensory nerves.

main headings: (1) the control of involuntary muscles of the more important organs of the body, particularly those of the heart and blood vessels, organs of respiration, lungs and bronchi, abdominal viscera, of the eye and of the skin (erection of hair, emotional blushing, etc.); and (2) the control of all secretory glands of the body both having external or internal secretions. Further, according to the peculiarity of function and distribution of the fibres the autonomic nervous system has been divided into two distinct systems—the sympathetic system and the parasympathetic system. These two systems show peculiar differences in their anatomical, physiological, and pharmacological reactions and are very often antagonistic to each other. These two systems send nerve fibres to the same organs but for opposite purposes, *e.g.*, the sympathetic dilates the pupil and accelarates the heart while the parasympathetic contracts the pupil and slows the heart. It will be convenient to describe the functions of the two systems and their differences when put in a tabular form in which it will be noted that the combined effect of the sympathetic and parasympathetic reactions leads to the normal balance of the functions of the different organs.

Table showing the functions of autonomic nervous system including the difference and antagonism between the sympathetic and parasympathetic systems.

	SYMPATHETIC	PARASYMPATHETIC
	Anatomically:	
21 22 23	Extends along the 8th cervical to the 2nd lumbar segments of the spinal cord. The fibres arise from spinal cord and end into a ganglion of the sympathetic chain.	Fibres arise from mid-brain, medulla and the sacral region in the cord, ending in some of the outlying ganglion *e.g.*, the ciliary ganglion of the eye and the ganglia found in the plexuses round the heart, the pelvic organs etc.
24		Mainly controlled by the brain.
25	*Physiologically:* Mainly controlled by spinal cord.	
26	Controls skin, erection of hair, (goose skin), skin glands (secretion of sweat), blood vessels of the trunk and limbs (vasoconstriction), pupil (dilatation), ciliary muscles, orbital muscles, lacrymal, mucous and salivary glands, sebaceous and sweat glands, accelaration of heart, thyroid gland, lungs, bronchi (dilatation), stomach and intestine, kidneys, pelvic viscera (contraction of sphincter of anus and vagina), liver glycogen converted into glucose; spleen discharges red blood cells.	Contraction of pupil, lachrymal glands, salivary glands, mucosa of the tongue; controls heart, stomach and intestines.
27	*Pharmacologically:* Particularly sensitive to adrenalin.	Particularly sensitive to atropine.
	The antagonism is shown thus:	
	Pupil—dilates	Contracts
	Heart—Accelerates	Slows
	Arterioles—contract	inhibit vasoconstriction
	Peristalsis—inhibited	Stimulates intestinal movements.
	Bladder—fills by contraction of sphincter and inhibition of detrusor.	Empties the bladder by inhibiting the sphincter and stimulating the detrusor.

The general physiology of the autonomic nervous system can thus be grouped under 4 main heads:—

(1) Movements of heart, lungs and intestines.
(2) Secretion of glands.
(3) Vasomotor impulses.
(4) Regulation of sphincters.

After describing the whole range of the nervous system of the body the attention may now be directed to the study of *Vayu* and its relation with the nervous system. This classification of the nervous sytem into (1) the cerebrospinal system which regulates the mental functions, function of the special senses, all locomotions and all sensation of the body; and (2) the autonomic nervous system which regulates circulation, respiration, secretions, etc., may be put as the headings in the following table and attempt may be made to put the qualities and symptoms enumerated above in the passages quoted from the *Samhitas* under these heads.

The table shows the classification of the 313 symptoms collected from the *Samhitas* under 9 groups into which all the manifestations of the nervous system are divided in terms of modern physiology. These 9 groups with their sub-groups are indicated in the second column of the table.,The groups numbered 1, 2, 4, 8 and 9 are those of the central nervous system and those numbered 3 5, 6 and 7 belong to the autonomic nervous system. The table shows further, that this modern physiological classification agrees essentially with the classification of *Vayu* under 5 forms. Thus Groups 1, 2, and 3 indicate *Prana Vayu;* group 4, *Udan Vayu;* group 5, *Samana Vayu;* group 6, *Apana Vayu* and groups 7, 8 and 9, *Vyana Vayu.* The sites of these 5 forms of *Vayu* are indicated in the Column No. 3.

From this table it appears that of the 313 symptoms due to normal and abnormal *Vayu, Vyan Vayu* represents 217 symptoms; *Prana Vayu* 57 symptoms; *Apan Vayu* 23 symptoms; *Saman Vayu* 11 symptoms and *Udan Vayu* 5 symptoms, making a total 313.

The maximum number of symptoms are due to pain—78 symptoms; muscular action 49 symptoms, secretion of sweat glands 28, mental symptoms 26, paralysis and wasting of muscles 25, contraction and dilatation of vessels 19; cardiac action, respiration and deglutition 19; excretion 17; disordered sensation 14; due to special senses 12 and other forms 26.

Five Kinds of 'Vayu'

This table shows that all the functions and 313 symptoms quoted above belong to one or other of the groups of the nervous system enumerated in the classification detailed above. This definitely proves that *Vayu* is nothing but the manifestations of the nervous system and nervous system alone.

From the above descriptions, based on gross anatomical findings on every dead body, one is forced to note the parallelism of the functions which exist between the autonomic nervous system and that of *Pran, Saman* and *Apan Vayus,* and that of the cerebrospinal system with *Pran* (mental function and special senses), *Vyan* and *Udan Vayus.*

The parallelism as indicated above is now dealt with under each *Vayu* separately.

Pran Vayu—The functions of *Pran Vayu* in normal condition are spitting, sneezing, belching, respiration, deglutition **ष्ठीवनक्षवतुद्गार श्वासाहारादि कर्म च।** to which Vagbhata adds **बुद्धीन्द्रिय हृदय मनोधमनी धारण।** intelligence, the action of the heart, mind and vessels (A.H.S.,20) and **वायुर्योवक्त्र-सञ्चारी सप्राणोनाम देहधृक्। सोऽन्नंप्रवेशयत्यन्तः प्राणांश्चाप्यवलम्बते।** (S.N. 1|7). Comparing these functions with those mentioned in tht first three groups of the table one finds the close relationship existing between them. In fact, all of the functions attributed to *Pran Vayu* are admirably the same when considered in the light detailed above. It can be remembered here that *Pran Vayu* is responsible for the function of the heart itself and the big vessels originating from it and not the circulation through the arterioles and capillaries. The capillary circulation regulated by the vasomotor system throughout the whole body maintaining the circulation of blood and also the lymph carrying the *Rasa Dhatu* to each cell is maintained by *Vyan Vayu* as shown in group 7 of the table. The organs of special senses, excluding those of the eye (vision) and skin (sensation) are the function of *Pran Vayu.* The skin sensation is controlled by the *Vyan Vayu,* while that of vision is not mentioned; probably this is included under *Alocaka Pitta.* As one **बुद्धीन्द्रिय** it is undoubtedly included under *Pran Vayu* according to Vagbhata.

Regarding the site of *Pran Vayu,* it is, again, equally remarkable. Caraka mentions the site **स्थानं प्राणस्य शीर्षोरः कण्ठजिह्वास्य नासिका** C.C. 28|4 *i.e.,* the head, thorax, ears, tongue, mouth and the nose. Compairing this with the brain, special sense organs and the cervical ganglia of the autonomic nervous system having their seat in the head, neck and thorax as shown in the first 3 groups of the table, a complete agreement between them is found. The first 3 groups include mental functions, special sense organs and those regulated by superior and inferior cervical ganglia of the sympathetic. It is to be particularly noted that while brain is not specifically mentioned as the seat of *Vayu,* the head has repeatedly been mentioned. Further, *Shira* is undoubtedly used synonymously as brain when we consider the definition of *Shira* by Caraka. Caraka mentions that the site of the life of living beings, the site of 5 *Jnanendriyas* and that which is the best part of the body, is known as *Shira.*

प्राणाः प्राणभृतां यत्र स्थिताः सर्व्वेन्द्रियानि च।
यदुत्तमाङ्गमङ्गानां शिरस्तदभिधीयते॥ C. S. 17|5.

As already mentioned, the function of *Pran Vayu* as the agent for vision is not mentioned probably this being included under *Alocaka Pitta.* According to my conception of *Pran Vayu* as detailed above a complete agreement could have been reached with Ayurvedic literature had the reading of Caraka been **स्थानं प्राणस्य शीर्षोरः कण्ठ जिह्वाक्षि नासिकाः** including thereby the eyes under this group. Some authorities have actually quoted the passages mentioning **जिह्वाक्षि नासिकाः।**

Saman Vayu—The function and site of *Saman Vayu* have been mentioned by Caraka (C. C. 28|4)—**स्वेददोषाम्बुवाहानि स्रोतांसि समधिष्ठितः अग्नेरन्तिकेश्य पार्श्वस्थ्यः समानोऽनिलबलप्रदः**

TABLE SHOWING THE CLASSIFICATION OF 313 SYMPTOMS COLLECTED FROM THE 'SAMHITAS' UNDER NINE GROUPS WITH SUB-GROUPS

		Site	Five kinds of *Vayu* C.C. 28/4	*Vayu* affections according to site S.N. 1/21-23	One *Vayu* covered by another C.C. 28/67, 68	Symptoms of abnormal *Vayu* C.S. 20/10	Abnormal *Vayu* A.H.S. 21/46-47	Abnormal *Vayu* C.C. 28/8	Abnormal *Vayu* according to site C.C. 8/22-34	80 symptoms of abnormal *Vayu* C.S. 20/9.	No. of symptoms	Total symptoms under each of 9 groups	Total symptoms under each kind of *Vayu*
PRANA VAYU	Group 1.												
	Mental defects	Brain	1	2	5	—	—	1	—	4	13		
	Nervousness and vertigo.		—	—	2	—	1	2	2	2	9		
	Disordered sleep		—	—	1	—	—	1	1	1	4		
												26	
	Group 2.												
	Organ of sight	Brain	—	—	—	—	—	—	—	—	0		
	Organ of hearing		—	1	1	—	—	—	1	3	6		
	Organ of smell		—	—	—	—	—	—	—	2	2		
	Organ of taste		—	—	—	1	1	—	—	2	4		
												12	
	Group 3.												
	Cardiac action	Superior & inferior cervical ganglion	—	1	1	—	—	—	1	2	5		
	Respiration		1	—	3	—	—	—	2	—	6		
	Deglutition		4	—	2	—	—	—	1	1	8		
												19	57
UDANA VAYU	Group 4.												
	Speech	Chest and throat	1	—	2	—	—	—	—	2	5		
												5	5
SAMANA VAYU	Group 5.												
	Digestion	Solar plexus, semilunar & superior mesentric ganglia	—	—	3	—	—	—	2	—	5		
	Other digestive disturbances		—	—	2	—	—	—	4	—	6		
												11	11
APANA VAYU	Group 6.												
	Excretion	Inferior mesenteric ganglion	1	2	4	—	2	—	6	2	17		
	Function of bladder, uterus and genital organs		1	2	—	—	—	1	2	—	6		
												23	23
VYANA VAYU	Group 7.												
	Contraction of vessels	Vasomotor and pilomotor nerves	—	1	—	—	2	1	3	—	7		
	Blushing.		1	4	—	1	2	—	3	1	12		
	Erection of hair		—	—	1	1	1	1	—	—	4		
	Secretion of sweat glands		—	3	3	7	2	—	6	7	28	51	
	Group 8.		—	13	4	4	6	6	22	23	78		
	Pain Disordered sensation	Sensory nerves	—	2	2	1	2	1	2	4	14		
												92	
	Group 9.		6	3	2	7	3	5	8	15	49		
	Muscular action		—	1	3	1	1	1	2	8	17		
	Paralysis	Motor nerves	—	1	—	—	1	1	4	1	8		
	Wasting of muscles		—	—	—	—	—	—	—	—	—	74	217
			16	36	41	23	24	21	72	80	313	313	313

Saman Vayu, the giver of energy, seated by the side of the digestive fire regulates the channels which carry the waste materials, the *dosas* and the fluids, and Susruta (S. N. 1|9)—*Samana Vayu* attached to the digestive fire and flowing through the stomach and intestines digests food, separates the products of digestion, one from the other and moves the waste products.

आमपक्वाशयचरः समानो वह्निसंगतः ।
सोऽन्नं पचति तज्जाश्च विशेषान्विविनक्ति हि ॥

The solar plexus, semilunar and superior mesenteric ganglia detailed in the group 5 of the table, representing *Saman Vayu* show exactly all the above functions and also that of the site. The chief functions of *Saman Vayu* are digestion due to vascular and secretory activity of the stomach and small intestines. Ileocaecal sphincter, kidneys and spleen are also controlled by *Saman Vayu* thus performing **भुक्तं गृह्णाति पचति विवेचयति मुञ्चति ।**

Apan Vayu—The function and site of *Apan Vayu* are detailed as follows:—

वृषणौ वस्तिमेढ्रञ्च नाभ्यूरू वङ्क्षणौगुदम् ।
अपानस्थानं यत्रस्थः शुक्रमूत्रशकृत्क्रियः ।
सृजत्यात्तं वगर्भौ च युक्ताः स्थानस्थिताश्च ते ।
स्वकर्म्म कुर्व्वते देहो धार्य्यते रैरणामयः ॥ C. C. 28|4.

The seats of *Apana Vayu* are the testicles, penis, bladder, umbilicus, thighs, hips and the rectum whence it performs the function of excretion of semen, urine, feces, menstrual blood and fetus.

Further—

पक्वाधानालयोऽपानः काले कर्षति चाप्ययम् ।
समीरण शकृन्मूत्र-शुक्र-गर्भांस्त्वान्यधः ॥

Being seated in the rectum, *Apana Vayu*, brings down in proper time, the wind, feces, urine, semen, fetus and menstrual blood.

One finds a complete corroboration of the functions and site of the inferior mesenteric ganglion mentioned in group 6 of the table with those mentioned by Caraka and Susruta quoted above. The excretory functions of *Apan Vayu* are performed by motor and inhibitory fibres to bladder, rectum, uterus and vagina. Although the sphincters are controlled by the autonomic nervous system, the acts of micturition defecation and parturition require a great coordination of voluntary action of *Vyan Vayu*—in the form of contractions of the abdominal and other muscles which constitutes the bearing down pressure.

Vyan Vayu—The functions and site of *Vyan Vayu* are:—

देहं व्याप्नोति सर्व्वस्तु व्यानः शीघ्रगति नृणांम् ।
गति प्रसरणाक्षेप निमेषादिक्रियः सदा । C. C. 28|3.

कृत्स्नदेहचरो व्यानो रससंवहनोद्यतः ।
स्वेदासृकश्रावनश्चापि पञ्चधा चेष्टयत्यपि ॥ S. N. 1|10.

These include mainly—movements, flexion, extension, closing and opening of the eyelids, regulation of the flow of sweat and blood, and circulation of blood, lymph and chyle throughout the body. The functions detailed under the groups 7, 8 and 9 *i.e.*, all motor, sensory and vasomotor fibres of the whole body indicate all the items quoted above. Moreover, along with normal movements, any disturbance or loss of movement *e.g.*, paralysis, incoordination, disordered gait, muscular rigidity, tremor, muscular atrophy will invariably follow when there is any disorder of *Vyan Vayu*. Susruta mentions the regulation of sweating as one of the functions. This includes, thereby, although not definitely mentioned, the erection of hair or blushing of the skin as these functions as well as sweating are the function of the pilomotor and vasomotor functions regulated by the autonomic system.

In the passages quoted above there is no mention of the mechanism of sensation regulated by *Vayu* not to speak of the *Vyan Vayu*. This must be included as function of *Vyan Vayu* as shown in group 8 (sensory) of the above table. Although the mechanism of sensation as the function of *Vayu* is nowhere mentioned, still it has undoubtedly been in mind when abnormality of Vayu is considered. This comes to the prominence when we consider the table which shows that 92 symptoms out of 313 due to disturbance of sensation which alone constitutes 30% of all symptoms due to abnormal *Vayu*. I have noted elsewhere that the most important indication of abnormal Vayu, rather to indicate in one word the derangement of *Vayu*, is characteristically **शूल** (Pain) which is not the effect of *Pitta* and *Kapha* while that of *Pitta* is **दाह** (burning sensation) and of *Kapha* **शोथ** (*Oedema*). This brings the function of *Vayu* in regulating sensation to the forefront and hence it is adequately included in the table as the function of *Vyan Vayu*. Caraka, however, mentions that *Vayu* has the prominent function in giving life and sensation. **"वायु प्राणसंज्ञा प्रदानां श्रेष्ठः** C. S. 25. *Vyan Vayu*, like *Pran Vayu*, thus represents both the cerebrospinal and the autonomic nervous system. All locomotions and sensations, both normal and abnormal are controlled by the cerebrospinal system while capillary circulation, sweating and blushing (by vasomotor fibres) and erection of hair (by pilomotor fibres) are regulated by the autonomic nervous system.

Udan Vayu—*Udan Vayu* has got one particular function and that of speech including singing, and the site is adequately mentioned to be **नाभि, उरः** and **कण्ठ—ऊरः** (the lungs) and **कण्ठ** (particularly meaning the larynx). These are indicated by the following passages:—

उदानस्स पुनः स्थानं नाभ्यूरः कण्ठ एव च ।
वाक्प्रवृत्तिः प्रयत्नोर्जोवलवर्णादि कर्म्मच ॥ C. C. 28|3.

The seat of *Udana Vayu* is umbilicus, chest and throat. The functions are speech, efforts, strength etc.

उदानो नाम यस्तूर्द्ध मुपैति पवनोत्तमः ।
तेन भाषित गीतादि विशेषोऽभिप्रवर्त्तते ॥ S. N. 1|8.

Udana Vayu seated high up regulates speech, singing etc.

There is thus a close agreement with the functions and site mentioned in Ayurveda to those mentioned in the group 4 of the table.

The function of the *Udan Vayu* which is a complex one may now be discussed in detail. For the purpose of speech several factors are called into play. (1) The lungs are called in to act as the bellow, rather the bag of the bagpipe so that expiration of either a uniform or varying character is maintained. This is often supplied by the contraction of other muscles of the thorax and the abdomen. (2) The larynx acts as the reed. This is done by the contraction or relaxation of the muscles of the two vocal cords, one on each side, so as to make the opening bigger or smaller. The more the contraction of the vocal cords *i.e.*, the narrower the opening the higher the note produced as in singing. By difference in contraction of the different muscles of the larynx thereby changing the tension and length of the cords the pitch, loudness, quality and other characters of the voice are produced. Loudness mainly depends on the force of the blast of air from the chest passing through the larynx. (3) Cavities of the mouth, (particularly modification in its shape), nose, nasal sinuses, trachea etc. play the chief role in producing the quality or timbre of the voice. (4) Muscles of the tongue, lips, throat, and mouth come to produce the vowel and consonant sounds *i.e.*, the articulation of speech. The shape of oral cavity plays an important role. Function of speech requires coordination and synchronisation of the muscles for functioning the (1) lungs, (2) larynx, (3) mouth, pharynx and nose. Nearly all of them are innervated by the cerebrospinal nerves. Of these, acting on the lungs are of spinal in origin while those of the mouth and larynx from the brain,—vagus (10th). spinal accessary (11th), hypoglossal (12th), and the lips by the facial (7th cranial) nerves. The trigeminal (5th) may also functionate in changing the size of the mouth. It is thus seen that the 7th, 10th, and 12th cranial nerves play the most important role in the mechanism of speech.

Functions of Normal *Vayu*

This study of the normal functions of *Vayu* forces immediately to the conclusion that all the diseases characterised by the disorders of the mind, intellect and nervous functions are caused by derangement of *Vayu*—**(वायुविकृति)** Further as nerve fibres exist in every minute part of the body so there exists no part of the body free from *Vayu*.

These nerves, after all, as the electric wires are, only the conducting material for the nerve impulses. *Vayu* like electricity is manifested by its functions and cannot be seen. It is thus appropriately called **अव्यक्त (अदृश्य मूर्त्ति) व्यक्तकर्मा च ।** The functions of *Vayu* as enumerated above apparently manifest that *Vayu* is possessing independant action **(स्वतन्त्र),** self-acting **(नित्य),** all pervading **(सर्व्वग),** guiding principal of *Dosas* **(दोषानां नेता),** originators of disease **(रोगसमूहराट),** quick in action **(आशुकारी),** always ready to act **(मुहुश्चारी),** capable of conjoint action **(योगवाही),** etc. Further due to the same function *Vayu* acts as **तन्त्रयन्त्रधरः, प्रवर्त्तकश्चेष्टानामुच्चावचानां.....** etc. (C. S. 12|8). Some discrepancy arises regarding the chief seat of *Vayu*. Caraka mentions **वस्तिः पुरीषाधानां कटिः सकथिनी पादावस्थिनी च वातस्थानानि, अत्रापि पक्काशयो विशेषेन वातस्थानम्।** (C. S. 20|6) that the urinary bladder, large intestines, pelvis, thighs, legs and the bones are the seats of *Vayu* of which *Pakkashya* (colon) is the chief seat.

The *Pakkashaya* is located below the navel. *Saman Vayu* is seated at the navel and as there is no other *Vayu* at a lower level than the navel other than the *Apan Vayu* Caraka apparently means that *Apan Vayu* is the chief *Vayu* according to the passage quoted above. This ignors the importance of *Pran Vayu*. From other passages it appears that *Pran Vayu* is the chief *Vayu* and it should be rightly so as this controls the circulation and respiration, the two most important functions of our body. Chest, the seat of *Pran Vayu*, should under this circumstance be the chief seat of *Vayu*. That *pakkashaya* is the chief seat of *Vayu* is further corroborated by Susruta and Vagbhata—**तत्र समासेन वातः श्रोनीगुदसंश्रय। तदुपव्यधोनाभेः पक्वाशयः** In brief, the seat of *Vayu* is between "sroni" and rectum and colon. (S. S. 21|6)—**स्थानं वातस्य तत्रापिपक्वाधानं विशेषतः।** AH. S. 12|2. In addition to this incongruity, it further becomes curious when it is observed that the brain and spinal cord have not at all been mentioned in Ayurvedic literature which are undoubtedly the main seat of *Vayu*. That cerebrospinal and the autonomic nervous system are the chief structures has undoubtedly been recognised by the *Tantric cult* who made a special contribution to the autonomic nervous system. The brain as the *Sahasrara* is the final and chief seat of all knowledge and function. According to *Tantric* literature *Muladhara*, the seat of which is near *pakkashaya* may be regarded to be the starting point of all energies and functions. In that sense *pakkashaya* as the chief seat of *Vayu* may be accommodated but this must be the starting point of activity and not the chief seat.

The cogent reason to ascribe *Pakkashaya* as the chief seat of *Vayu* appears to be on therapeutic ground. Enemas, purgatives and emetics are the main agents for purification of body in cases of deranged *Dosa*. Of these agents, enemas are the best for mitigation of Vayu. In the words of Caraka—**वाते आस्थापनानुवासनं—पक्काशयमनुप्रविश्य केवलं वैकारिकं वातमूलं छिनत्ति, तत्रावजिते वाते शरीरान्तर्गतवातविकारः प्रशान्निमापद्यन्ते—यथा वनस्पतेर्मुले छिन्ने स्कन्धशाखावरहकुसुमफलपलासदीनां नियतो विनाशस्तद्वत्।**

C.S. 20|12. That is, enemas in derangement of *Vayu*—The enema entering into the colon destroys the root of any aggravated *Vayu*, and thereby mitigating *Vayu* in this region mitigates all aggravated *Vayu* throughout the body, just as by cutting at the root of a tree, the trunk, branches, twigs, leaves, flowers, fruits etc. are all destroyed.

In this sense Pakkashaya (colon) may be regarded to be the chief seat of *Vayu*.

From what has been detailed under each of the five *Vayus*, a grouping can now be attempted to show the general function of normal *Vayu*. This has been admirably summarised by Susrata **प्रस्पन्दनोद्वहन पूरण विवेक धारण लक्षणो वायु पञ्चधा प्रविभक्त शरोरं धारयति** (S. S. 15|3). Respiration and other movements of the body **(प्रस्पन्दन),** circulation **(उद्वहन)** nutrition maintained by taking in of the food, its digestion, assimilation and supply of the same to every part of the body **(पूरण),** separation of all waste materials from the body and their excretion **(विवेक),** and maintainance of the body **(धारण),**

Each of these functions is that of each of the 5 *Vayus* detailed above. Further Caraka mentions **उत्साहोच्छ्वास निश्वास चेष्टाधातुगतिः समा। समो मोक्षागतिमतां वायो कर्म्मविकारजम्॥** (C. S. 18|42). This again includes the same functions *e.g.*, effort, respiration, proper action of mind and body, proper flow of *Dhatu* and proper excretion. All the functions enumerated in the passages **वायुस्तन्त्रयन्त्रधरः** etc.—and other passages quoted above are most adequately represented in the respective groups as shown in the table which at once shows each function being regulated by the respective *Vayu*.

Function of Abnormal Vayu

In the table showing the classification of the different forms of *Vayu* in which the various symptoms and functions have been arranged under respective headings a rich material mainly dealing with the manifestation obtained as a result of derangement of *Vayu*, are indicated. This rich material is gathered from the symptomatology of only a few **वातजव्यधि।** A scrutiny of other **वातजव्यधि** not considered here *e.g.*, **वातजशिरोरोग, वातज हृद्रोग** etc. will further show that they also manifest a striking analogy to those already mentioned.

Thus the theory that *Vayu* is nothing but the manifestation of the nervous system is well established.

Properties of Vayu & Vayu Temperament

In all the *Samhitas* **वायोः स्वरूपं,** Properties of *Vayu*, is mentioned at the begining followed by other characters of *Vayu i.e.*, seat, function etc. The importance thus given to properties of *Vayu* is very often misleading to the beginner, at least that had been so to the present writer. A great stress has been given to the character of *Vayu* which constitute **रूक्ष शीतो लघुः सूक्ष्मश्चलोऽथविशदः खरः।** (C. S. 12|3)
This at once indicates that one is speaking of a tangible object which is rough to touch etc. A beginner is puzzled to compare this to the other character of *Vayu* particularly **अमूर्त्तित्व, सूक्ष्मत्य** and **अव्यक्त।** Only on minute scrutiny it can be gathered that these characters are not those of *Vayu* itself but of the persons having preponderence of *Vayu*, i.e., of *Vayu* temperament. This point may now be discussed in detail.

It has repeatedly been mentioned in all Ayurvedic literature that it is the equilibrium of the three *Dosas—Vayu, Pitta* and *Kapha* which maintains the normal body and also all its normal functions. The temperament and other qualities of the mind and body originate and remain normal so long the equilibrium of the *Tridosa* is maintained. Such an individual might be called of having **समप्रकृति** indicating that there is no abnormal prominence of any particular *Dosa*. This condition is termed variously as **प्रकृति, स्वभाव, स्वरूप** etc. Any abnormal prominence of particular *Dosa* will lead to a series of manifestations, peculiar and constant according to the *Dosa* affected. This abnormality may be either congenital or acquired. Congenital when the abnormality takes place along the growth of the fertilized ovum. Caraka says **यस्य यस्यावयवस्य बीजे बीजभाग उपतप्तो भवन्ति, तस्य तस्यावयवस्य विकृतरूप यायते, नोपयायते चानुपतापात।** C. Sa. 3|25.
It becomes acquired due to persistent use of food in abnormal quantities and qualities having stimulating or depressing effect of some particular *Dosa*. (Vide C. B. 6|7). It has further been shown that the congenital form of abnormal temperament, growing simultaneously along with the cells of the body, so alters them that the same temperament persists throughout the life and that without any change. It occurs thus—**समप्रकृतिर्वातप्रकृतिर्नभवति, वातप्रकृतिः पित्तप्रकृतिर्नभवति सयप्रकृतिर्वा।** C. S. 7|40. *Vayu* controls the personality and temperament of an individual by influencing the following qualities.

People differ in many respects both mentally and physically according to the state of the nervous system. The temperament depends on the stability of the nervous equilibrium of the individual. It is of common occurrence to find two individuals doing the same sort of work, say reading a novel or engaged in a conversation on a serious topic and some children playing in the room. A shout from a child or even their presence will be annoying to one of the persons and the shout will at once set his mental equilibrium disturbed and probably he will punish the child or drive out all of them. The other individual, on the other hand, will probably pay no heed to the noise of the children, even a child jumping on his person will not be able to provoke any irritation in him. In the former individual the nervous system is in a state of high sensitiveness so that a slight stimulus sets in the whole mechanism in action. This might, again, be compared to a mob which is in a highly agitated condition, a slight stimulus which will not attract any notice to another group of individuals, say those of an excursion party, will provoke the excited mob to usher in a disaster. Persons of a *Vayu* temperament, similarly, have got their nervous system in tension and according to the group of the nervous system affected will show the series of manifestations as detailed in Ayurvedic literature.

It has been noted that the functions of *Vayu* can be divided under 3 groups: (1) acting on the muscles for the purpose of movements of all forms throughout the body, (2) acting on the glands mainly on the skin (sweat and sebaceous) and of the intestines (digestive secretions), (3) acting on the mind. According to the preponderance of one or more of the above 3 groups of functions affected, the individual of *Vayu* temperament will manifest 3 main groups of effects. First, when the nerves influencing on the muscular system are in highly sensitive condition the person will show hyperactivity of the muscular system as described by Caraka—all the movements, efforts, taking meals and all actions are light, swift, agile. There is also restlessness in joint, eye, eyebrow, jaw, lips, tongue, head, shoulders and limbs. He is also talkative.,These functions have been attributed by Caraka to be produced by lightness **(लघुत्व)** motility **(चलत्व)** and part of multiplicity **(बहुत्व)** properties of *Vayu*. This is shown by the following passage: **वातस्तु रूक्षो लघु चलः बहुः..... भवन्ति।** C—B—8/84.

The vasomotor nerves being in a state of irritation will lead to constriction of the vessels of the skin as what takes

place during the cold stage of an attack of malaria and so the individual is always getting shivering and will not like cold. This is due to coldness **(शीतत्व)** property of *Vayu* as mentioned by Caraka quoted above. The relaxation of the veins leads to their prominence **(बहुकाण्डरा शिरा प्रतानाः)**. Due to the second factor, *i.e.*, influencing the secretions in which the secretions particularly those of the skin are diminished, a diminished secretion of the sweat and sebaceous gland leads to roughness of the skin and its appendages, *i.e.*, hair, nail, etc. The absence of the sebum leads to cracks and fissure of the skin. Diminished quantity of secretion producing dryness of the throat and larynx will lead to a rough, low-pitched, hoarse and indistinct voice. All these have been attributed by Caraka to be due to the dryness **(रूक्षत्व)** roughness **(पारुष्य)** and non-slippery character **(वैशद्य)** of *Vayu*. These have been well described in the above passages quoted from Caraka.

Thirdly, increased influence on the mental functions, leads to quick excitement of the physical and mental inclination, also disappointment. The individuals quickly get fear, anger and aversion. They can grasp any problem quickly but forget equally quickly. This is produced by the property of quickness **(शीघ्रत्व)** of *Vayu*.

It requires further study to explain how an individual of *Vayu* temperament becomes lean and short in stature, how he gets less strength, or less longivity, less children, less vitality and less wealth as detailed by Caraka, and also how he becomes dishonest, jealous, thievish, ungrateful and unsteady in friendship and also possessing character like goats, jackals, hares, rats, camels, dogs, vultures, crows and asses as described by Susruta.

Causes for the Derangement of Vayu

The causes mentioned in the *Samhitas* are quite definite and all lead to either hyperstimulation or depression of the nervous system. They are: (1) fighting with a stronger individual, (2) excessive exercise, (3) sexual excess, (4) excessive study with loud reading, (5) fall, (6) excessive running, (7) excessive massage of the body, (8) excessive jumping, (9) sudden jumping, (10) excessive swimming, (11) keeping nights, (12) carrying excessive load, (13) constant riding on horse, elephant or carriage, (14) excessive walking, (15) excessive eating of various substances which influence *Vayu*, (16) fasting, (17) irregularity of meals regarding quantity, quality and time, (18) intake of food before proper digestion of previous meal, (19) forcible restraint on the action of sphincters as well as of sneezing, cough, vomiting, etc. **तत्र बलवद्विग्रहार्तिव्यायाम ..... प्रकोपमापद्यते।** S. S. 21/23-24.

It appears that all the above causes are nothing but over-exertion and hence overstimulation of the nervous system. The only item "fasting" may act as a depressent to the nervous system.

Many of the above factors have been mentioned as causes of **वायुन्नीना** It appears that all these act by hyperstimulation followed by depression of the nervous system.

(*Continued from page 182*)

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JOURNAL OF THE

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CALCUTTA, APRIL, 1943

DEATH IN WAR TIME

With the bombs falling on our cities, the problem of death and injury due to enemy action has become important for everybody. And the problem is interesting to a medical man for two reasons, as he shall have to face the problem both as a citizen and as a physician. He as a citizen must know how his bodily remains will be treated after his demise, his earthly belongings adjusted and his relations provided for. As a physician he must know how much help he can render at the time of an accident.

In such cases, the first problem obviously is diagnosis of death. One must know that sufficient provision has been made to diagnose death by a man who is well qualified to do so. Of coure, we are sure that *all* deaths will be diagnosed only by qualified medical men. Cases are not rare, where death has been diagnosed when there was suspended animation only. All standard works on medical jurisprudence record quite a number of such 'deaths'. The Bhowal Sannyasi case can be cited as an example. What is true for ordinary ailments, is much more true for air raids, as shock and other complicating injuries will be quite in abundance during a raid. It is sometimes very difficult to differentiate properly between death and shock. At least a medical man is required to do justice to these borderline cases.

Furthermore, if a medical man is on attendance to every site of air raid mild or intense he will at least be able to sort out dead bodies from those who require further observation (for example head injury cases), cases which require immediate attention on the spot, hysterical cases and so on. This procedure ensures two things—firstly, that those who could be saved will be saved, *secondly unscrupulous* persons will not be able to do away with borderline cases. The plan that "the police will arrange for collecting as soon as possible from streets and buildings all dead bodies for whose disposal no arrangement is being made by private parties" is fraught with many difficulties and probably to obviate a part of such difficulty the Central Government has passed the following amendment * to War Injuries Ordinance.

> "The person managing any dispensary or hospital shall, if so required by the Central or a Provincial Government by general or special order,—
>
> Provide at the dispensary or hospital, medical and surgical treatment for persons who have sustained injuries of the nature specified in sub-section (1) of section 3, (air raids).

* Ordinance, Vol. I of 1942. Published by the 'Gazette of India', Jan 1, 1942.

And there is no reason why all medical men who come under these amendments should not be acquainted with their duties immediately and the question of remuneration settled. If the physician is to charge the patients for his attendance he is bound to give a certificate to the party. But certain persons are entitled to have pension and compensation, in case they are injured by enemy action. A physician must know in what form he shall have to issue a certificate. We are quite in the dark what we are expected to do even when the enemy is knocking at our very door. Proper co-operation between the Government machinery and the medical bodies like Indian Medical Association to settle the details is urgently indicated.

This, however, is not the whole story. A death is always attended with various difficulties, of which the question of identification is a very important one. Unless the dead person is properly identified death cannot be finally fixed on that person nor can his properties, insurances, etc., administered. The Government notice that "the public will also be warned that it is in their interest that they should carry identity discs or labels showing (*i*) name and sex, (*ii*) father's/husband's name, (*iii*) address and (*iv*) name and address of the next of kin. Government will accept no responsibility for arranging to establish identity of air raid victims found without such discs or labels on their persons" is not ideal one because an identification disc may be damaged, lost or misused. Necessarily there will be either chaos or injustice done if we are to pin our faith implicitly on the identity disc. To avoid these difficulties the authorities in Britain have adopted a different course. We heartily commend them. Kitchen expresses them in the following words:

> "Now, however, if a registrar has reason to believe that a person has or may have died in consequence of war operation, and that his body has been destroyed, or that it cannot be found, recovered, or identified, or has been buried under another name or as the body of a person unknown, he may report to the coroner accordingly. The coroner then, unless he thinks to do so would be useless, has a notice exhibited in an appropriate place—*e.g.*, a local newspaper—stating the name and address of the vanished person, that his death is believed to have occurred in the district on a certain date, that an enquiry is to be held at a given time and place, and that any persons who know anything about the matter should inform the coroner or attend the enquiry.
>
> He holds the enquiry privately and without a jury, and does not have any body exhumed. If he then forms the opinion that the person has died from war operations, he issues a certificate to that effect, and this is equivalent to the leave of the High Court to presume the death."

Insurance difficulties shall have to be faced also. Insurance companies do not for obvious reasons pay their dues unless all the documents are in order. We are not told whether the death certificates issued by the Municipal Corporation will be binding to the insurance companies also.

This is for big cities. But what about villages? Many villages have suffered air raids and must have had many difficulties in this direction. When one thinks of India, he must also think of its villages.

In England the following arrangements have been made: "To meet the large number of deaths caused by enemy action, the authorities have considerably modified for the duration of the war the ordinary duties of the coroner. To begin with, deaths due to war operations are taken out of the hands of the coroner altogether unless they are specially referred to him. If an authorised person, commonly the clerk of the country borough council, or of the council of a London Metropolitan borough, certifies that a body is that of a person who has died in consequence of war operation, then the local authority may have the body buried or allow the relatives to dispose of it. The registrar's certificate or coroner's order is not necessary. The coroner need not be informed of a death which is reasonably believed to be due to war operations, nor need he, if he is satisfied that this is the cause of the death, take any action. The registrar must be informed by any person who would otherwise have to report the death to him; therefore, a medical practitioner who was attending the deceased at the time of his death must send the registrar a death certificate. If this certificate satisfies the registrar that the death was due to war operations, he will register the death without reporting it to the coroner. Otherwise, he gives the informant (under the 1926 Registration Act a relative or the person in charge of the house or present at the death an opportunity to call in a medical practitioner to inspect the body and give a medical certificate, or, if a certificate has already been given, to obtain from a certifying doctor a more explicit certificate. If the informant refuses, or if no medical certificate is produced to satisfy the registrar that the death was directly due to war operations, the registrar will report to the coroner that the death was alleged to be due to war operations. Then, if the coroner decides that the death was due to war operations, he so informs the registrar but does not hold an inquest. If he decides that the death was unnatural and not due to war operations, he holds an inquest in the ordinary way.

"A doctor who attended the deceased person before his death should issue the ordinary certificate unless he knows that the clerk to the council or the commanding officer (if the deceased was in one of the Services) has certified that death was due to war operations. In certifying any form of violence as a cause, he should be careful to add the words 'war operations', if he is satisfied of their truth. He does not then report the death to the coroner. If a doctor is called in by the relatives after death, he may use the ordinary form of certificate for a war death, though he would not be allowed to use it for a death due to any other cause. He should modify the certificate by taking out the words "last seen alive by me" and the words "not seen", so that the statement indicates that he saw the deceased after death and not before. The registrar will not accept an informant's statement that death was due to war operations unless it is supported by a medical certificate; but no certificate is necessary for a death certified by the clerk to the council as a war death.

"The rules governing cremation are also modified. If the applicant for cremation can produce to the medical referee of the crematorium a registrar's certificate showing that the death was due to war operations, he need not also produce the usual certificates by two doctors, the authorized pathologist, or the coroner. He must, however, obtain a written authority from the medical referee before the body can be cremated. A 'body' under these rules includes part of a body. Similar provisions are made for Scotland.

"The coroner is also given a new and unusual power. Under war conditions, when persons killed in air raids are often never seen again, the need arises unusually often to presume the death of a missing person so that his property may be administered. In ordinary times leave to presume death is given by a judge of the Probate Division, and the proceedings may cost up to £200 and take several months. Now, however, if a registrar has reason to believe that a person has or may have died in consequence of war operations, and that his body has been destroyed, or that it cannot be found, recovered, or identified, or has been burried under another name or as the body of a person unknown, he may report to the coroner accordingly. The coroner then, unless he thinks to do so would be useless, has a notice exhibited in an appropriate place —*e.g.*, a local newspaper—stating the name and address of the vanished person, that his death is believed to have occurred in the district on a certain date, that an inquiry is to be held at a given time and place, and that any persons who know anything about the matter should inform the coroner or attend the inquiry.

"He holds the inquiry privately and without a jury, and does not have any body exhumed. If he then forms the opinion that the person has died from war operations, he issues a certificate to that effect, and this is equivalent to the leave of the High Court to presume the death."*

This clear-cut scheme shows clearly how vague all schemes on the subject in India are. We suggest that (*i*) all air raid casualties must be attended by a physician at the site of the raid and who will diagnose death or otherwise and issue relevent certificates and Police will only be responsible for disposal of dead bodies. (*ii*) Identification of all dead persons should be established before the disposal of dead bodies, if possible. In case of difficulty a medical man may be requisitioned for. (*iii*) Notices should be given in local newspapers regarding missing people for identification and a central body should take over the operations for proper administration so that the party can have them when identity or claim is properly established.

REPRESENTATION ON THE BOARD OF MANAGEMENT OF THE KING GEORGE'S AND ASSOCIATED HOSPITALS

General Buckley, a senior officer of the I.M.S., carried out an investigation into the affairs of the King George Medical College Hospitals in Lucknow for 2 months in 1941. This group of Hospitals is unique that it is under the administrative control of the Executive Committee of the University of Lucknow. The main theme of General Buckley's report is that the administrative control of the Hospital should be transferred to the Provincial Government and not be left to the University. From a careful perusal of the published report of General Buckley, we are not convinced that grave reasons exist for this change. The irregularities and shortcomings under the present administration pointed out by General Buckley are capable of rectification easily by any administrative authority. Deficiencies

* Medical Annual, 1942, p. 218 and. 219.

can be found in most institutions official or non-official, if looked for. Public memory in spite of its proverbial shortness may still remember a similar case of one of the premier Government Hospitals in India a few years back. The change recommended is really a change from one group of men (University) to another group (Government officials). The only common denominator is the I.G.C.H. who is in both the bodies but he may be a lone voice among the other 22 Executive Councillors of the University whereas under Government control he is the responsible head of the department, and he may have a greater voice in the destinies of the Hospitals if the transfer takes place.

We hear that the Advisory Board of Management of the Hospitals is being reconstituted and the only representative of the Lucknow Medical Association has not been given a place in it though such a representation has been continuously allowed in the past and General Buckley's Report has nothing to say against such a representation of a large section of local practitioners. This policy of ignoring the unwritten privilege of an important group of well-wishers and supporters of the local institution is extremely deplorable. If this is the first step towards the improvement of the institution, we could hardly advise the Lucknow Medical Association "to burn a few gallons of petrol" in trying to keep a place for themselves under the sun of the Hospital Advisory Board—probably under benevolent Government control. Even though the Hospital Advisory Board may have no executive powers as hitherto, a sprinkling of outspoken members is beneficial in a majority "Yes man" group. We can only express a pious hope that better counsel will prevail and a representative of the Lucknow Medical Association will be given a seat in the newly formed body.

We await the decision of H. E. the Governor as to the ultimate controlling authority of this group of Hospitals, an institution of which all medical men in U.P. should be proud.

(*Continued from page 196*)

be avoided. Masks, with a layer of paper or other impervious material between the layers of gauze, must be worn by all those taking part in dressings. Fingers must never be allowed to come in contact with the dressing, the wound, or the skin in its neighbourhood. All manipulations must be done with instruments, or the dresser must wear gloves. Bedclothes, the chief source of dust, may be heavily contaminated, and in order to diminish dust infection, dressings should not be done until at least an hour has elapsed since bed-making or sweeping the ward, and all draughts must be excluded. Sterlizing instruments, lotions, dressings, and the wound itself should be exposed to the air only when necessary, and they should be kept covered at all other times. Great care must be exercised in attending to the details of sterilization of instruments and utensils, in cleansing baths, in changing plasters, and in exposing wounds in x-ray or massage departments in order to prevent hospital infection. In this connection it is noteworthy that too much reliance is often placed upon surgical spirit, which is not a trustworthy disinfectant. If secondary infection is avoided healing will proceed normally provided that the general nutrition and the supply of vitamins, especially vitamin C, is maintained. The local application of growth-promoting substances is unnecessary, but skin grafting should be undertaken early if the original injury was associated with skin loss.

OBSTETRICS AND GYNAECOLOGY

Observations on the Aetiology and Prophylaxis of Prothrombin Deficiency and Haemorrhagic Disease in the New-Born*

Macpherson (*J. Obstet. Gynæc. Brit. Emp.*, 1942:49:368) considers various conditions of pregnancy and labour (pregnancy toxæmia, premature delivery, dietary deficiency of foods rich in vitamin K, prolonged labour) in cases seen at the Royal Infirmary, Edinburgh, in relation to the degree and duration of hypoprothrombinæmia in the new-born. Of these conditions, none was found to have any constant effect upon the prothrombin index of the new-born except deficient diet during pregnancy. The children of mothers who gave a history of such dietary deficiency during pregnancy were found to show uniformly low values for the prothrombin index. Prolonged labour *per se* did not adversely affect the prothrombin index in the new-born. By contrast, when prolonged labour was complicated by repeated interference under chloroform-anæsthesia, the prothrombin index was found to be so low even at birth as to imply that prothrombin synthesis in the fœtal liver had been depressed by the action of chloroform. The author suggests that chloroform may be toxic to the fœtus *in utero* in a similar way and in the same circumstances as in the mother. The evidence obtained from feeding 4 hydrocephalic babies on an absolutely vitamin K-deficient diet from the time of birth indicates that conditions of intake and absorption of vitamin K in the new-born do not affect the initial fall in the prothrombin index but that they influence the subsequent recovery towards normal. It appears, moreover, that in the absence of a dietary source, vitamin K cannot be synthesized by the intestinal flora and absorbed by the intestinal mucosa in sufficient quantities to maintain the level of the prothrombin index. The author reports in detail 15 cases of hæmorrhagic diseases of the new-born. In 2 of these cases, bleeding occurred as a complication of erythroblastosis fœtalis (icterus gravis). In every case of simple hæmorrhagic disease in which the antenatal diet was investigated, there had been a grossly deficient intake by the mother of foods with a high vitamin K content. The incidence of other features of pregnancy and labour was quite irregular. The most frequent site of bleeding was gastrointestinal, and it is suggested that this may be related to the hyperacidity of the fasting juice in the new-born. Cerebral hæmorrhage was proved to have occurred in 4 of the 15 cases of hæmorrhagic disease. Two of these cases were in very premature babies. In every case, symptoms indicated that the effect of birth trauma had been accentuated by the hypoprothrominæmia.

The author concludes that it should be possible to prevent hæmorrhagic disease of the new-born by proper supervision of the mother's diet during pregnancy. When this has not been feasible, the same end can be achieved by adequate administration of vitamin K to the mother late in pregnancy or during labour.

Abstracts marked with an asterisk are received through courtesy of the British Information Service, 3 Hanover Street, London, W.I.

CURRENT MEDICAL LITERATURE

MEDICINE

Non-Articular Rheumatism in the Army: Symptomatology, Aetiology, Treatment*

Hutchison (*Glasgow M. J.*, 1942:19:33) does not claim to present any new facts or figures; he does, however, give a clear and concise account of a disability which frequently requires the attention of the Army Medical Officer. The non-articular rheumatism which is discussed is divided into fibrositis and sciatica, 77 per cent of the author's 60 cases coming under the former heading. In discussing the clinical features of fibrositis, he holds that there are three predominant objective signs—well-defined tenderness, spasm, and nodule formation. He requires at least two of these for the diagnosis to be made. Symptoms are vaguer and consisting as they do of pain and stiffness, lay open the path for the malingerer. True sciatica is a less common but more serious condition, and may be differentiated from sciatic pain due to fibrositis by absence or diminution of the ankle jerk, wasting and hypotonia, and occasionally analgesia. In discussing ætiology, the author admits that any suggestions regarding it must at present be speculative. Five possible causes are given, and more than one may be present in the same case. In the treatment of these cases rest and physiotherapy are claimed to be most effective. The use of injections of a local anæsthetic is extensively discussed and the exact technique is given; the author feels, however, that too much has been claimed for this method of treatment, and he has found that the use of such injections does not do away with the need for physiotherapy. All of his 60 patients with non-articular rheumatism returned to duty, the average time in hospital for the cases of fibrositis being 20 days, and for sciatica 39. He agrees that the cases of sciatica in this series were mild, and that the condition may lead to prolonged invalidism.

Epidemic Myositis with Neuritis, Erythema and Meningeal Symptoms*

Williams (*Quart. J. Med.*, 1942:10:283) under this title records five cases occurring in recruits of the R.A.F. from the same station and within the period April to July, 1940. All had recently been inoculated with tetanus toxoid and T.A.B. vaccine, but thousands of other recruits similarly inoculated did not develop any symptoms. All these patients were sent to hospital with the tentative diagnosis of cerebro-spinal fever. The constant clinical features were acute febrile onset with pain and extreme tenderness in the muscles, especially the proximal groups of the limbs. Symptoms of meningitis were present in all cases, neck stiffness and Kernig's sign being well marked in two and slight in the others. The cerebrospinal fluid in one case showed 400 cells, of which half were polymorphonuclear, half lymphocytes. In two other cases there was a slight pleocytosis which included polymorphonuclears. Two of the patients complained of tingling at the periphery of the limbs. The deep reflexes were in no case lost, but were diminished in three. In three cases an erythematous rash was present, followed in two by desquamation. In the case with 400 cells per cmm. in the cerebrospinal fluid, optic neuritis was observed. These cases were considered to form a homogeneous group, probably of virus origin. The inoculations may have acted as a precipitant of the illness. The possible relationships to epidemic myalgia (Bornholm disease), dermato-neuro-myositis, and acute lymphocytic chorio-meningitis are discussed, and reference is made to a report by Pattison (*J. Roy. Army Med. Corps.*, 1940:75:388), of a somewhat similar group of cases occurring at the same time under the diagnosis of cerebral influenza.

Paravertebral Abscess with Rupture into the Pleura or Lung*

Brooks (*Brit. J. Tuberc.*, 1942:36:49) writes that a few isolated instances of transpleural rupture of a paravertebral abscess arising as a consequence of vertebral or mediastinal disease have been reported in the literature. The complication is usually thought to be quite rare. As a member of the staff of the Brompton Hospital (London), he encountered six cases within the space of three years, and he concludes that the striking nature of the intrathoracic sequelæ of such a rupture, the relatively undramatic and frequently slight symptomatology of spinal caries, and the technical difficulties in making a complete diagnosis have combined to obscure the diagnosis in many such cases. Of the six cases reported in the present paper (full case histories are given) five were due to tuberculous and one to actinomycotic spinal caries. Three cases were undergoing treatment for their known spinal lesion when rupture occurred, and in the three others spinal caries with paravertebral abscess was entirely unsuspected. When penetration into the lung occurred, the patients died following more or less acute pulmonary suppuration. The prognosis was better in the two cases in which only the parietal pleura was penetrated. Tomography, or the delineation of the sinus with lipiodol, were of great assistance in establishing the diagnosis with certainty. Analysis of the clinical histories showed that in every case prodromal symptoms of pleural irritation (unproductive cough and pleural pain) antedated rupture of the abscess by days and sometimes by weeks. Therefore, knowledge of the syndrome, and the occurrence of premonitory symptoms permit the application of proper therapy with the object of averting catastrophe. Repeated aspiration of the abscess (or its pleural extension) may be sufficient, or if this seems likely to fail, free surgical drainage is essential and should always be combined with immobilisation of the spine by a frame or plaster. If rupture into the lung has already taken place, the patient, after surgical drainage of the abscess, should be placed in a posture on his frame so that the bronchial fistula is uppermost as shown by lipiodol. In such cases, although the outlook is very grave, a plastic operation may assist in closing the fistula.

A Simple Test for Headache

Scott (*J. Nerv. & Ment. Dis.*, 1942:96:64) writes that there is often considerable difficulty in distinguishing functional headaches from those due to organic disease, and the physician will welcome any assistance in differentiating them. The test which the author proposes has given good results in cases of

brain tumour, hypertension, meningitis, cerebral syphilis, anxiety neurosis and malingering. The test is simple, merely consisting in firm pressure with the thumbs in the malar regions for about ten seconds, after which the pressure is suddenly released. In the case of irreversible headaches of organic origin no change takes place, but in the vast majority of patients suffering from hysteria, anxiety or malingering there is a temporary relief from the headache, which suddenly returns when the pressure is released. The same result occurs on repeated trial. Under proper conditions the same effect may be secured by pressure on any other part of the body, but for various reasons the malar pressure has been considered the most suitable.

The Preservation of Museum Specimens in War-time*

Wentworth (*J. Path. & Bact.*, 1942:54:137) writes that the preservation of museum specimens is of great importance at ordinary times, for permanent record and for teaching purposes. At present, under conditions of general war, the need is all the greater because pathological lesions may be met with which may not occur again. Nevertheless the usual preservatives and mounting fluids, glycerine and spirit, are scarce and difficult to come by in adequate quantity, if obtainable at all for such a purpose. Hence the value of the method here described, which dispenses with both of these fluids. The author devised it in pre-war days, has used it for four years and finds it gives entire satisfaction. The natural colours of the specimen are quickly restored (within 48 hours or so in many cases), owing to the reducing properties of the sodium hydrosulphite ($Na_2S_2O_4$), not, be it noted, thiosulphate or hyposulphite ($Na_2S_2O_3$). Moreover the amount to be used should not exceed that mentioned, or softening of the tissues will result and the fluid become discoloured.

The preparation of the fluid and the method of its use are thus described: "(1) The specimen to be mounted should be fixed for at least three months in either formol-saline or the following mixture: Sodium acetate 40 gm., formaldehyde (40% solution) 100 c.c. and water 100 c.c. Neutralization is unnesessary and undesirable. (2) It is then immersed for at least a fortnight in a 10 per cent solution of sodium acetate to remove the excess of formalin. (3) After being trimmed, resurfaced and weighed, the specimen is mounted in a 10 per cent watery solution of sodium acetate from which the air has been expelled by boiling for a minute or two or by a suction pump. Sodium hydrosulphite is then added to the fluid in amount equivalent to 2·5 g. per 1,000 g. of the tissue to be preserved and the lid immediately sealed. As soon as it can be done with safety, the jar should be gently rocked to ensure an even diffusion of the hydrosulphite. The growth of moulds can be prevented by the addition of 1·5 c.c. of 80 per cent phenol or 1 g. of camphor to each litre of mounting fluid. Brown discolouration of the fluid from oxidation of the phenol is prevented by the hydrosulphite."

SURGERY

Sepsis in War Wounds of the Limbs*

Ross (*Brit. M. J.*, 1942:1:589) in the present paper reviews this subject in the light of recent experience.

The conditions prevailing in most war wounds favour bacterial invasion, and are unfavourable for tissue repair It is a matter of experience, however, that wounds often heal better than would be anticipated from these conditions, and the explanation of this discrepancy lies to a great extent in the differentiation between contamination and infection. The presence of micro-organisms on the surface is inevitable in war wounds though virulent organisms are rarely found in the early stages. This surface contamination can be cleansed by excising the wound at the earliest possible moment. The introduction of sulphanilamide into the wound after excision may be beneficial, and is harmless provided it is not used as an alternative to thorough excision. Only under exceptional circumstances will it be permissible to suture wounds completely even at this early stage. Excision means removal of dead or damaged tissue, and should be carried out with due care to preserve all healthy tissue, and especially important structures, such as nerves and tendons. If conditions are favourable for the development of gas gangrene—compound fractures, lacerated wounds of muscle contaminated by brick dust, debris, or soil, and punctured wounds through clothing—particularly if there is some unavoidable delay in excising the wound, a prophylactic dose of gas-gangrene antitoxin should be given. This may be either 7,500 International Units of Cl. Welchii antitoxin alone, or a mixture of antitoxins for Cl. Welchii (3000 International Units), Cl. Septique (1,500 International Units) and Cl. Oedematiens (1,000 International Units). Prophylactic use of antitoxin implies the intramuscular injection of serum at once and not waiting until the wound looks suspicious.

Wounds in which infection is already established when they are first seen should not be excised, and as the organisms will have penetrated deeply into the tissues the surface application of sulphonamides is of little value, though oral administration is useful. The wound should be laid open so that all foreign material may be found and removed, and free drainage may be established. Strips of vaselined gauze laid lightly between the wound edge make a good dressing, and the limb should then be immobilized in plaster. The advantages of this method are not only that it rests the part and is comfortable, but also that by preventing œdema it reduces lymphatic absorption, and it also obviates the risks of frequent dressings. The importance of secondary (hospital) infection can scarcely be overemphasized. Most of the wounds which after a few days or weeks in hospital are heavily infected with virulent organisms—in particular hæmolytic streptococcus and staphylococcus pyogenes—were free from this infection on admission, and it has been demonstrated that these organisms have usually entered the wounds from the hands or throats of the attendants or from the air of the ward when dressings were done. Experience has shown not only that these secondary infections occur frequently, but also that they have serious effects in delaying healing, causing local spread of inflammation, and producing outbreaks of tonsillitis and "unexplained" pyrexia and ill health both in patients and ward staff. Every effort must, therefore, be made to block the channels of this kind of infection.

Recent investigations in Britain have shown the following precautions to be imperative if secondary wound infection is to

(*Continued at the foot of the page 194*)

NOTES AND NEWS

NON-PHARMACOPOEIAL DRUGS

The following circular and questionnaire dated 31st January, 1943, was issued by the Government of India—Commerce Dept. under the signature of Dr. B. Mukerji, D.Sc., M.D., F.A.Ph.S., Secretary, Advisory Panel on Drugs and Medicines, Biochemical Standardisation Laboratory, (Government of India) on the subject—Import Trade Control, Advisory Panel of Drugs and Medicines. The attention of all our members is drawn to the importance of the questionnaire. Relevant replies to the whole or any portion of it may be sent to the Secretary, Indian Medical Association by the 30th June, 1943.

Dear Sir/Sirs, You are perhaps aware that the Government of India have for sometime been considering the question of revising the present method of control of imports of drugs and medicines from abroad to ensure that only the most suitable medicines and in the most suitable quantities are imported under the Import Trade Control Scheme. With this object, they have set up an "Advisory Panel on Drugs and Medicines" with the following terms of reference:—

"To advise the Government of India as to which Drugs and Medicines, other than those the formulas of which are included in the Official Pharmacopœias of the exporting countries, it is essential to import into India from abroad.

(Note:—The term 'Official Pharmacopœias' includes the British Pharmacopœia, the British Pharmaceutical Codex, the United States Pharmacopœia, and the National Formulary of the United States of America).

2. The Panel met at New Delhi on 10th December, 1942, and decided to issue a set of Questionnaire to appropriate parties in order to collect representative opinion regarding the drugs and medicines which should be imported into India.

3. The Panel desires to have the views of all persons, associations or bodies interested in this question with respect to the points mentioned in the terms of reference. It will feel grateful if you will kindly go through the questionnaire and send your replies, under the groups suggested, as early as possible. It is not contemplated that all questions are to be answered by each and every one to whom the questionnaire is being issued. It would be helpful if answers are given against those questions only which are in your field.

4. It is needless to point out that the question of supply of drugs and medicines is of paramount importance to national health and well-being. Steps to improve the present shortage of important remedies in India can only be taken if the Panel is able to submit its report early. This will depend largely on the help and co-operation which you will extend to us.

5. Replies to the questionnaire and all other communications may please be addressed to the Secretary, Advisory Panel on Drugs and Medicines, at the above address so as to reach him before the 10th March, 1943.

QUESTIONNAIRE

I. What drugs or medicines (outside those included in the B.P., B.P.C., U.S.P. & N.F.) are normally imported and distributed or sold by you?

Kindly send your answers under the following headings:
Proprietary name
Name of manufacturer and/or country of origin
Composition, if known
Uses
Quantity and value of drugs imported annually
Pre-war figure
Present figure

N.B.—It will help in the collation of the opinions obtained if answers are given according to the scheme laid down below:

1. Vitamin preparations.
2. General & nerve tonics including tonic wines.
3. Laxatives, purgatives, etc.
4. Analgesics, sedatives & hypnotics.
5. Antianæmic remedies including liver and iron preparations.
6. Diagnostic Agents: (*a*) For liver and gall bladder, (*b*) Lungs, (*c*) Kidneys, (*d*) Stomach & intestines
7. Remedies for stomach & bowel troubles, including digestives, antacids and antidiarrhœal remedies.
8. Heart tonics and other cardio-vascular remedies.
9. Hæmostatics (internal & external)
10. Cough remedies including anti-asthmatics.
11. Remedies for liver and gall-bladder troubles.
12. Specific remedies: (*a*) anti-syphilitics, (*b*) anti-dysenterics, (*c*) anti-malarials, (*d*) anti-bacterials, (*e*) anti-tuberculosis.
13. Uterine and other drugs for women.
14. Gland products.
15. Antiseptics & disinfectants including genito-urinary antiseptics.
16. Injectable products
17. External Remedies, e.g. liniments, plasters, ointments, poultices, inhalations, etc.
18. Miscellaneous, including (*a*) Throat remedies, (*b*) Germicidal soaps etc.
19. Any other drugs or remedies not covered in the above list.

II. Which of the above items you consider absolutely essential from the point of view of their utility to the medical profession or the public?

III. Are you manufacturing any products which may be used as equivalents or substitutes for any drugs and medicines normally imported? Do you consider that you can supply the normal demands of India? If not, can you give some idea regarding your manufacturing capacity.

IV. In view of the acute shortage of shipping space, would you suggest any reduction or even total stoppage of the importation of any particular drug or medicine?

V. What is your opinion regarding the replacement of any of these items by locally produced equivalents or substitutes? Please indicate such items.

N.B.—Kindly write your answers on a separate sheet and put in your Signature thereto.

BOOK REVIEWS

THE MEDICAL COLLEGE MAGAZINE, BENGAL—Vol. X, No. I, June, 1942. Published by the Medical College Students' Union, Calcutta.

We congratulate the Literary Secretary of the Medical College Magazine, Bengal, whose name is not given any prominence anywhere for publishing such a well got-up magazine at this emergency period. The Medical College Magazine was not published for the last four years and we are happy that with its advent it could hold its position which it held before.

If one peruses the contents of the journal, one becomes convinced of one improvement in the present volume. More than half of the contributions are from the students, which is rather a deviation from the past custom of almost exclusively printing the teachers' papers.

The magazine does not give any report of any work in any of the departments except one on Ca, P, and Fe contents of leafy vegetables from a student of the 4th year class. So we are not aware of the amount of original work done in the institution. We hope to see more of original contributions as the clinical wealth of the institution and the possibilities of its various laboratories are indeed enormous.

R. OBADIAH.

DISEASES OF WOMEN—by Ten Teachers. Edited by Clifford White, Comyns Berkley and Frank Cock. Seventh Edition, 1942. Pages viii+435. Demy 8 vo. Illustrated. Published by Edward Arnold & Co., London.

This is a short text-book for students. It is clearly written and well illustrated. In this edition there has been a change of two authors. The arrangement of the chapters has been altered. Sulphonamides have been introduced into the treatment of the pelvic inflammatory conditions. The anatomy of the pelvic Organs and the physiology of menstruation have been revised, and a simpler classification of ovarian tumours and uterine polypi has been given. The book will prove useful to the students.

S. N. BAGCHI.

A HAND BOOK OF MODERN PHYSIOLOGY—by Drs. R. K. Pal and A. Chakravertty. 2nd Edition, 1942. Pages xvj+780. Demy 8 vo. Illustrated. Published by The Book Co. Ltd., Price Rs. 12-8.

A text book of Physiology for Indians by Indian authors was a real want and the volume under review removes that. It comes out at a time when foreign publications are not locally available and is thus doubly welcome.

The second edition is a thorough improvement over the first and the authors are to be congratulated for the care they have taken to make the volume up-to-date. The chapters on biochemistry and metabolism, endocrines and vitamins are comprehensive and cover almost all major recent works done in those directions. A note on applied physiology after each chapter is a welcome feature and it is desirable that the medical students will learn, during their study of physiology, such important matters as the liver function or kidney efficiency tests.

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OBITUARY

DR. R. N. BHATIA, F.R.C.S.

We regret to announce the death from heart failure of Dr. R. N. Bhatia, F.R.C.S., a famous surgeon of U. P., on 17-2-43 at his residence in Lucknow.

An old and senior member of the Lucknow Medical Association, he was for sometime Professor of Surgery in King George's Medical College, Lucknow, and was very popular amongst the general public as a surgeon of skill and eminence. His death is a great loss to the Association and the public.

The Lucknow Medical Association passed the following resolution at its meeting held on 25-2-43:

"The Lucknow Medical Association places on record its deep sense of loss and sorrow at the death of Rai Bahadur Dr. R. N. Bhatia, F.R.C.S., the well known and popular surgeon of Lucknow. In the passing away of Dr. Bhatia, the medical profession of these Provinces have lost a surgeon of great eminence. The loss of the surgeon of such great skill and experience will be equally felt by the public of these Provinces."

(*Continued from previous column*)

The appendix contains very useful informations specially with reference to Indian data of food values. We would have, however, welcomed a fuller treatment of the nervous system including the mechanism of maintenance of posture and equilibrium and recent conception regarding the functions of the cerebellum. We would also like to draw the attention of the authors to some loosely expressed sentences which appear incompatible in such a good compilation.

We have no hesitation in recommending the book to students of physiology in the medical colleges and in the undergraduate classes of the universities.

S. M. BANNERJI

THE COIMBATORE DISTRICT MEDICAL ASSOCIATION'S ANNUAL, No. 3, 1942. Price Rs. 2/-.

We are very impressed with No. 3 of the Coimbatore District Medical Association's Annual for 1942. We fully realise that it could not have been an easy job to publish such an Annual in these days. The Annual contains almost all the papers read at the monthly meetings of the Association and one of them is a striking original article by Lt. Col. P. V. Karamchandani, I.M.S. An extremely useful new line has been adopted that the Annual contains the names and addresses of the members of the Coimbatore District Medical Association and provides a complete guide to the medical institutions and their staff and also the complete list of registered medical practitioners of the district. The Annual Report shows the Branch to be in a flourishing condition and is a model which might be viewed with envy by most of the other branches of the I.M.A. If we were not concerned with the shortage of paper and financial stringency, we would have advocated that at least a reprint of the pages 130 to 150 should be sent to each and one of our branches as an example of what a branch can do. The Annual was distributed at the Annual Meeting held on 28-2-43 at Coimbatore and there was a dinner at night at the City Municipal Hall. We congratulate the branch on its efficiency.

S. C. CHATTERJEE.